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ON DISEASES

OF THE

LUNGS AND PLEURÆ

INCLUDING

TUBERCULOSIS AND MEDIASTINAL GROWTHS

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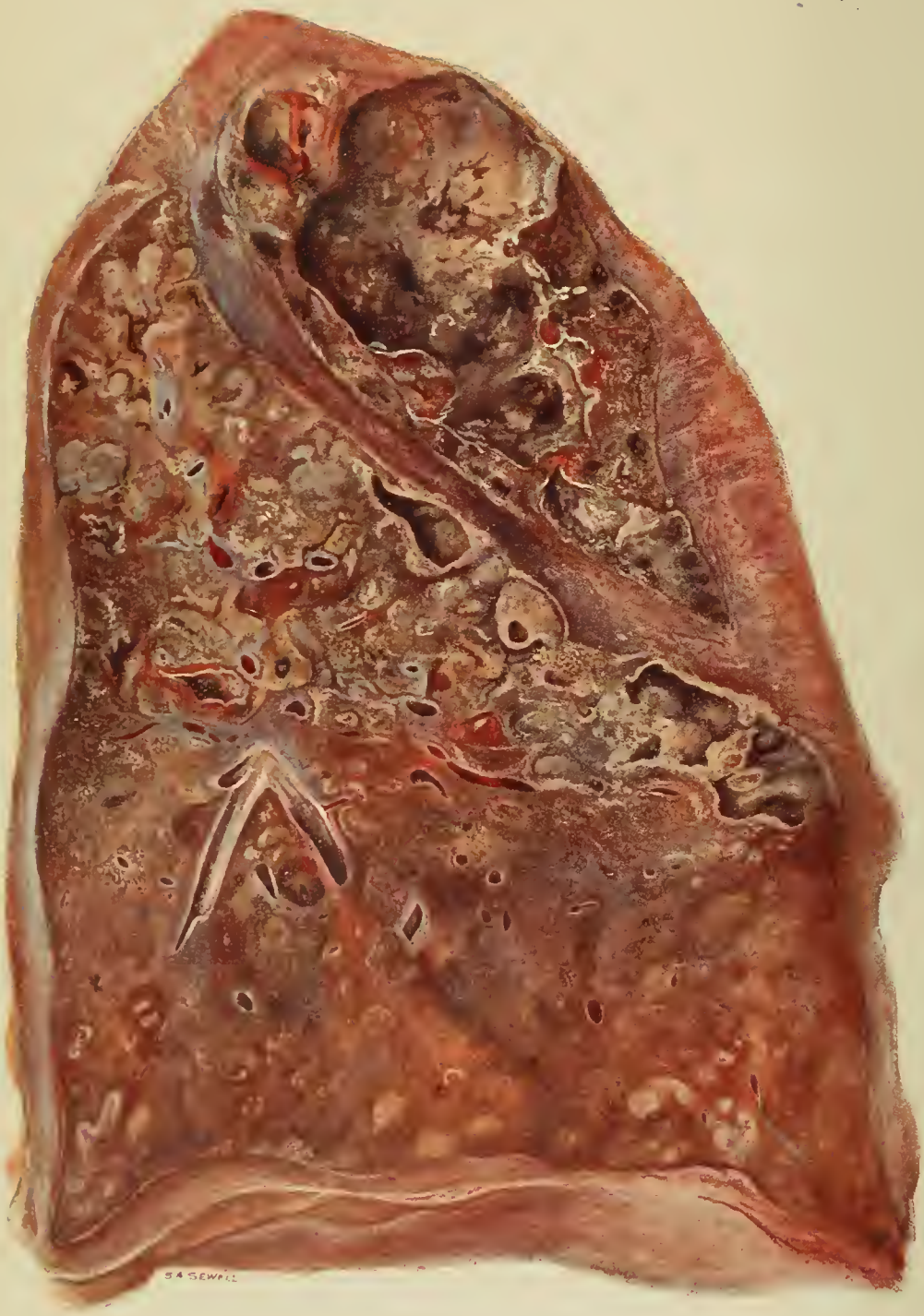

The Œdematous Pleura—a stage in the formation of the thickened Pleura.

THE ŒDEMATOUS PLEURA—A STAGE IN THE FORMATION OF THE THICKENED PLEURA

The specimen shows a section through the left lung. The upper lobe is much shrunken and shows advanced pulmonary tuberculosis, including a large cavity. Extensive tuberculous disease is also present in the lower lobe. Over the upper lobe and in the great fissure, the pleural layers are seen to be separated and the space crossed by fine vascular loops; the intervening meshes are filled with serous fluid, and the whole presents a gelatinous appearance. As yet the pleural membrane itself is hardly at all thickened. The dense fibrous thickening which results at a later stage may be seen in Plates XXX. and XXXI.

From a boy aged seventeen, who died from chronic pulmonary tuberculosis with terminal miliary tuberculosis of lungs and kidneys, and with tuberculous lesions in the larynx and intestines.

(From the Museum of the Brompton Hospital, $\frac{5}{6}$ natural size.)

PLATE XXVIII

ON DISEASES
OF THE
LUNGS AND PLEURÆ

INCLUDING

TUBERCULOSIS & MEDIASTINAL GROWTHS

BY

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SIXTH EDITION

WITH ILLUSTRATIONS

PHILADELPHIA
P. BLAKISTON'S SON & CO.
1921

Med.
21-14678

PRINTED IN ENGLAND

RC756
P87

PREFACE

TO SIXTH EDITION

ANOTHER nine years have elapsed since the last edition of this work and twenty-eight years since the appearance of the fourth edition.

In the course of this time the last word has been said on antiseptic methods in surgery and more tardily in medicine, methods upon which so much of modern treatment of pulmonary disease is based, and in still greater degree its prevention. During this period bacteriology has made great strides, and has succeeded in elucidating many problems dealing with pathological and therapeutic action. If in the latter respects, though rich in other fields of work, bacteriological research has been less fruitful in practical results in the field of lung diseases, it must be gratefully acknowledged that such investigations have served to deepen our insight into the pathology of these diseases and the problems of immunity, and have in no slight degree rendered more clear our outlook for future treatment.

As in recent editions, fairly full references to literature on the subjects dealt with have been appended to each chapter, but the authors have not hesitated to express as definitely as possible their own conclusions after due consideration of the views of others. Some of the older references have also been retained in view of historic interest and continuity.

Several new clinical cases have been introduced in illustration of some of the varieties of phthisis, and also of other diseases, but many of the old cases have been preserved as clinical pictures the outlines of which have not faded with time.

Reference to tuberculin has been considerably shortened

in acceptance of the lessened value attached to its use except in special cases. In prescribing this remedy the cubic millimetre has been adopted as the basis for dosage instead of the cubic centimetre as in former editions.

New chapters have been added on Gunshot Wounds of the Chest, Chylothorax, Massive Collapse of the Lung, Sporotrichosis, and Artificial Pneumothorax; and surgical methods have been more fully dealt with in connection with certain diseases.

The Authors have gratefully to acknowledge the help of Professor Bainbridge, Dr. H. M. Gordon, Dr. W. Jobson Horne, Dr. L. S. T. Burrell, and Professor Gask in revising the proofs on certain subjects and giving valuable criticism and suggestions thereupon.

Professor Karl Pearson and Dr. Brownlee have also favoured the Authors with valuable advice in connection with the ætiology of phthisis, and have revised and completed to date the diagrams illustrating its epidemiology and prevalence. Recent figures showing the standardised death-rate from phthisis have been courteously supplied by the Registrar-General.

The Authors' thanks are also due to the Medical Research Council for permission to reproduce drawings from their publications which are referred to in the text.

The Index of the volume has been somewhat shortened in the able hands of Miss A. Newbold, and finally the Authors have most gratefully to acknowledge the services of Miss E. F. Parry in revising and arranging the text of the whole book.

LONDON,
October, 1920.

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DISEASES OF THE LUNGS AND PLEURÆ

CHAPTER I

ON SOME ESSENTIAL POINTS IN THE ANATOMY AND FUNCTIONS OF THE LUNGS

It would seem a necessary preliminary to a discussion of the pathology, detection and treatment of chest diseases to recall to the recollection of the reader some of the more essential points respecting the anatomy and function of the lungs which he may have in part forgotten. Both physiology and anatomy, learned in the earlier stages of the student's curriculum, must be relearned as he advances to practical medicine, and oft reverted to in after years; and it is further true, as Addison asserted, that morbid anatomy and pathology need to be more nearly regarded as reflecting light upon the structure and functions of healthy organs. In the physiological laboratory we read life forwards, in the post-mortem room backwards; and in the latter we may perceive those instructive extensions and compensatory developments of healthy structure and function which have, perhaps, for long maintained the vital balance in otherwise hopeless cases.

It is impossible truly to comprehend the manifold incidents and conditions of such diseases as asthma, emphysema, pleuritic effusion, pneumothorax and many other chest maladies, without a working knowledge of those statical and dynamical conditions of respiration in health, to a consideration of which we will now without further apology proceed.

Anatomy.—The average weight of the lungs in the male may be taken as 24 ounces for the right, 22 ounces for the left; in the female about 5 ounces less in each case.

Perhaps one could not better summarise the rough anatomy of the lung than by quoting the first four aphorisms of Addison[1] respecting it. These affirm: (1) "That the aerial cellular tissue of the lungs is made up of well-defined rounded or oval lobules, united to each other by interlobular cellular membrane, each lobule constituting a sort of distinct lung in miniature, having its own separate artery and vein; (2) that these lobules do not communicate directly with each other; (3) that they do not consist of the globular extremities of as many bronchial tubes, but are made up of a collection of cells in which, by a common opening, a minute filiform bronchial tube abruptly terminates; (4) that the pulmonary artery accompanies the bronchi, branch for branch, to the minutest divisions of the latter."

The general arrangement of the bronchi is shown in the accompanying radiograph from Dr. Brünings'[2] work (Plate I.).

If we trace the bronchial tubes from the trachea into the depth of the lungs, we find that they divide in a peculiar manner: (1) from the main bronchi down to a calibre of $\frac{1}{6}$ inch (4 mm.) they divide symmetrically or dichotomously; (2) beyond $\frac{1}{6}$ inch the bronchi proceed in straight lines, gradually diminishing in size and giving off lateral branches alternately at an angle of about 45°; (3) these lateral branches again divide dichotomously until they reach a diameter of about $\frac{1}{100}$ inch (0·25 mm.), soon after which they terminate in several *alveolar passages* or *atria*, which, with the infundibula and air cells attached, form the lobules referred to by Addison. The mode of termination of the bronchioles is illustrated in the diagram of the lung lobule (Fig. 1), constructed by Professor W. F. Miller.[3] Whilst in general the branchlets of the bronchial tree have their direction onwards, some, according to Dr. Ewart,[4a] may be found to take a backward or recurrent direction to their termination in the pulmonary lobules. A distinct tendency may be observed throughout the bronchial system for each bifurcation to occur in a plane perpendicular to that of the preceding bifurcation, alternate bifurcations thus taking place in the same plane.[4b]

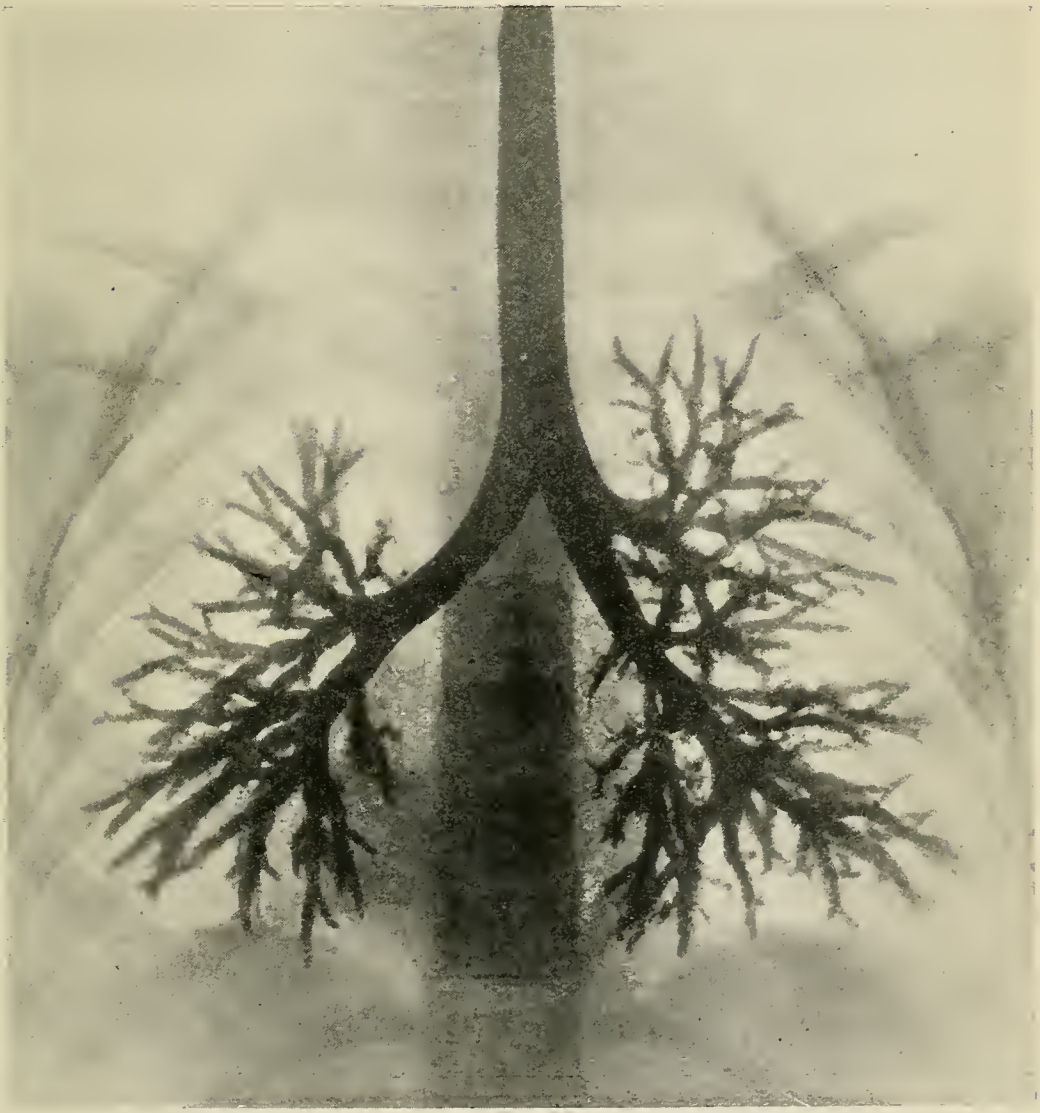
Down to a diameter of $\frac{1}{24}$ inch (1 mm.) the bronchial divisions are possessed of cartilages and muscular coat, are lined by a ciliated epithelium, and are furnished with mucous glands. Their arterial supply is thus far derived from the bronchial

PLATE I

X-RAY PHOTOGRAPH, TAKEN FROM BEHIND, SHOWING THE NORMAL DISTRIBUTION AND ARRANGEMENT OF THE BRONCHIAL TREE

From a cadaver in which the trachea and bronchi had been filled with a solution of lead oxide and gelatine.

PLATE I



X-Ray Photograph, showing the Normal Distribution and General Arrangement of the Bronchial Tree. (After Dr. W. Brünings.[2])

To face p. 2.

artery, the capillaries of which terminate in the bronchial veins, forming a part, therefore, of the *systemic* circulation.

Below the diameter of $\frac{1}{24}$ inch the bronchi have neither cartilages nor mucous glands, but their muscular coat and the elastic fibres persist, and ciliated epithelium lines the tube, although soon to give place to flattened non-ciliated cells. The arterial supply, still derived from the bronchial artery, terminates in the pulmonary venous capillaries, and forms therefore a part of the *pulmonary* circulation.

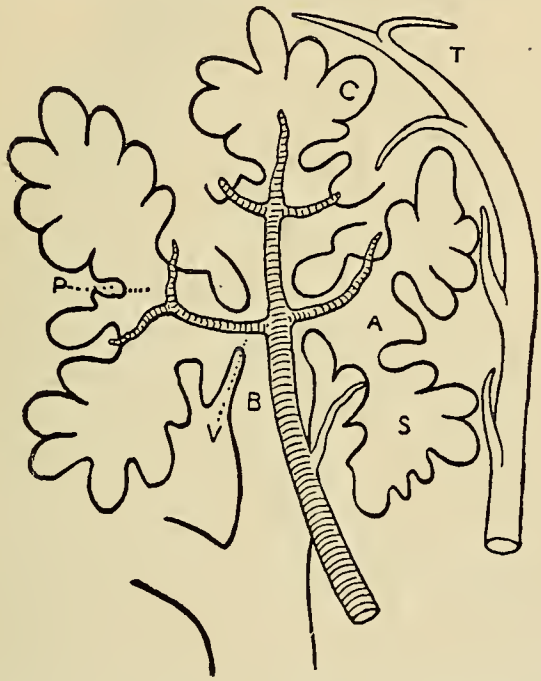


FIG. 1.—SCHEME OF THE LUNG LOBULE OF THE MAMMALIAN LUNG (AFTER PROF. W. S. MILLER, FROM SCHÄFER'S "ESSENTIALS OF HISTOLOGY").

B, terminal bronchiole; V, vestibule; A, atrium or alveolar passage; S, air-sac (infundibulum); C, air-cell (alveolus); P, ending of pulmonary arteriole; T, commencement of pulmonary venule.

The alveoli, or little recesses, about $\frac{1}{100}$ inch (0·25 mm.) in diameter, with which the alveolar passages and infundibula are studded, are composed mainly of a framework of fine fibrous tissue, among which numerous elastic fibres are to be seen. They are lined internally by a delicate flattened epithelium, and are surrounded externally by a network of capillaries.

Each lobule of the lung is thus made up of a lobular bronchus or bronchiole, with the alveolar passages, infundibula and alveoli connected with it. Adjacent lobules are separated from

each other by bands of fibrous tissue, forming the so-called interlobular septa, continuous on the one hand with the subpleural fibrous tissue, and on the other hand with that surrounding the bronchi and bloodvessels, and thus ultimately with the connective tissue of the mediastinum. On the surface of the lung in the adult, as may be seen in the accompany-

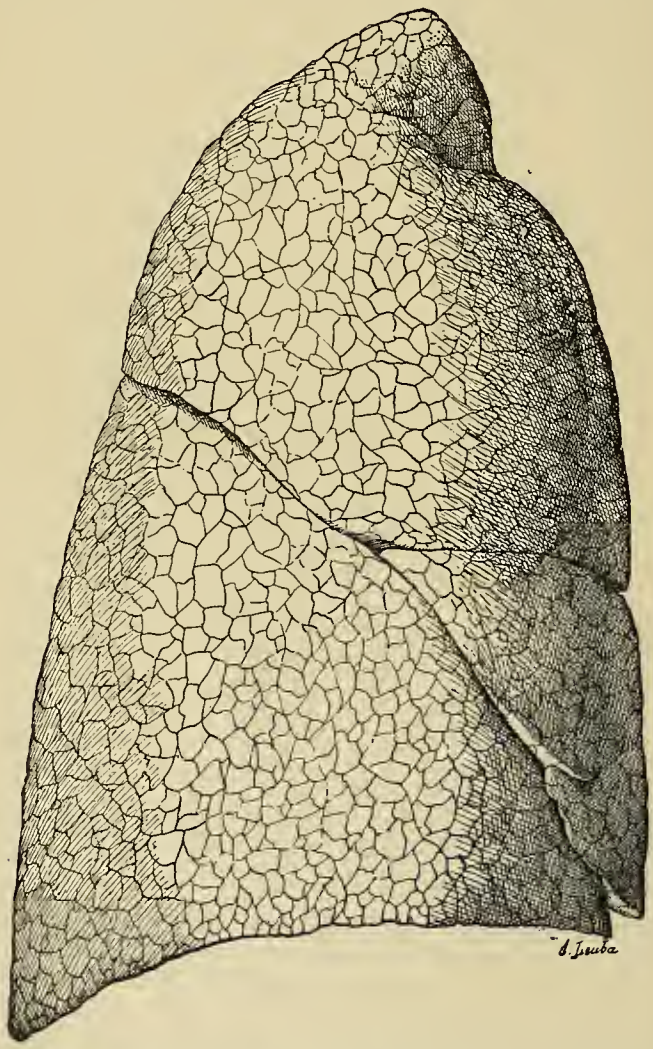


Fig. 2.—External Surface of the Right Lung, showing the Division into Lobules (after Poirier and Charpy[5]).

ing figure (Fig. 2), the division between the lobules is generally visible to the naked eye, their boundaries being marked out by a greater or less deposit of carbonaceous material. Their size varies, according to the late Professor MacAlister, from $\frac{1}{4}$ inch to $\frac{2}{5}$ inch (6 mm. to 10 mm.) in diameter.

With regard to the bloodvessels of the lungs, it should be

borne in mind that the pulmonary artery, carrying dark blood, subdivides with the bronchial tubes, and finally furnishes a capillary layer to each alveolus, each branch proceeding to its final distribution without communicating with any other branch. A junction between the pulmonary and systemic vessels is, however, affected at the pulmonary lobules by the discharge of the capillaries of the bronchial artery into those of the pulmonary vein as aforesaid. The veins have no valves, and for the most part take a separate course to the root of the lung in the interlobular or pulmonary fissures. They anastomose freely.

Respiratory Function and Mechanism.—In the recesses of the lungs within the alveoli and alveolar passages is effected that interchange of gases by means of which oxygen is on the one hand absorbed, and carbon dioxide (CO_2) exhaled, although the minutest bronchi also allow of a certain oxygenation of blood through their fine membrane. The laws under which this interchange is carried on are in the main those which regulate the diffusion of gases and their passage through moist membranes, though it is possible, as urged by Douglas and Haldane[6] and others, that when the need of oxygen is great, a direct secretory activity of the alveolar cells is brought into action, whereby oxygen is taken up by them and excreted into the blood of the pulmonary capillaries. The interchange of gases thus brought about is, so to speak, the basis of the respiratory function; but in order that this function may go on uninterruptedly, it is obvious that special provisions must be made for a regulated renewal of the blood operated upon and a thorough and sustained ventilation of the minute air chambers in which the process takes place. The rhythmic contractions of the right ventricle of the heart insure a continuous current of blood through the alveolar capillaries, and the rhythmic respiratory movements secure, under conditions to be immediately stated, a thorough ventilation of the lungs.

Let us now glance at the mechanism by which these respiratory movements are effected. If a small portion of the medulla—an area the limits of which have not been very clearly defined, but which may be described as lying below the vaso-motor centre in the immediate neighbourhood of the nuclei of the vagus nerves—be removed or injured, respiration

ceases, and death at once ensues. This portion of the nervous system was named by Fleurens the *nœud vital*, or ganglion of life. It is generally spoken of as the respiratory centre. The action of the centre in starting and regulating the respiratory function is partly automatic, partly the result of reflex stimulation. It is automatic in the sense that the fundamental respiratory impulses are generated in the centre itself as a result of the stimulating influence of increasing quantities of carbonic acid in the circulating blood; but reflex in that it is most delicately responsive to peripheral stimuli reaching it from diverse quarters.

It need scarcely be observed that in the fœtus at the full term the lungs are in a state of complete and airless collapse, that the diaphragm is in a position of extreme convexity upwards, and the thorax in the position of utmost contraction, the respiratory function being effected by the interchange of gases between the fœtal villi of the placenta and the maternal blood in which they are steeped.

In the new-born child, however, on the placental circulation being interrupted, carbon dioxide accumulates in the blood and stimulates the respiratory nervous centres, thus exciting acts of inspiration. This central nervous stimulation would probably not alone be sufficient, but it is supplemented, on the first exposure of the child to the external air, by a general excitation of the cutaneous nerves, causing strong inspiratory movements. Doubtless also the opening out of the air cells is further facilitated by a kind of erectile influence produced by the injection of their capillaries with blood, a view originally suggested by Dr. Philip Hensley,[7] in his Goulstonian Lectures for 1872, and supported later by the experiments of Liebermann.[8] As the result of these combined forces, air penetrates the lungs, and is never again fully expelled.

It is not at all curious that the lungs, thus once inflated, should remain more or less permanently expanded, retaining in their interior a certain amount of air. They, like any ordinary elastic air bags, would do this, whether in or out of the body. But in the body, both during life (within the limits of ordinary respiration) and after death, the lungs are maintained at a degree of expansion greater than that to which they would, by their mere elasticity, revert. This is a fact admitted by modern physiologists, but its importance

in clinical medicine is perhaps not yet sufficiently recognised. In order to account for the maintenance of the lung in this semi-expanded state, we must assume that when the muscles of respiration, which are for the most part inspiratory muscles, have once been excited to action, they remain permanently shortened by virtue of their vital contractility or *tonus*. And in the rapid course of tissue growth the ribs and cartilages become moulded to that wider arc which they have thus been brought to assume.

The residual tension or pressure depending upon the elasticity of the lungs has been measured by different observers with somewhat different results. Dr. Carson,[9] who in 1820 was the first to demonstrate the existence of this reserve tension, estimated it in different animals as equivalent to from 6 to 20, or more, inches of water, according to the size of the animal. He fully perceived the importance of this elastic force, both in the respiratory mechanism and as an aid to the circulation, though he misinterpreted its effects.

Donders,[10] in 1853, made some experiments on the human subject. By connecting a manometer with the trachea, and then carefully opening the thorax, he was able to ascertain the residual elastic tension of the lungs, and concluded that in the healthy person it was equal to 80 mm. (about 3 inches) of water. He further allowed 20 mm. for the tonicity of contractile elements in the lung, making thus during life a total pressure or traction of 100 mm. of water or 7·5 mm. of mercury.

Dr. Salter, in 1865,[11] found the residual tension in the dog equal to 4 inches of water.

Dr. M. Perls,[12] in 1869, gave the result of 100 experiments, conducted in the same manner as those of Donders, upon persons who had died of different diseases: out of these, in twenty-five cases death had been caused by diseases remote from the lungs, although in most instances the lungs were in some degree affected. The highest residual tension registered by Dr. Perls' manometer from these twenty-five cases was 60 mm. of water; the lowest, 5 mm.; mean, 35·3 mm. It is probable that the experiments of this observer give results somewhat short of those of perfect health. It is, at all events, noteworthy that the highest elastic pressure obtained by him in the twenty-five cases in which the lungs were presumably

healthy did not equal that (63 mm.) obtained from cases of bronchitis and phthisis respectively. The subject has not attracted much attention in recent years, but, pending further inquiry, we shall probably not be far wrong if we take a mean of the figures observed by Hutchinson and Aron[13] and those of Donders, and accept 5 mm. of mercury as a fairly accurate estimate of the pressure or traction exerted by the elasticity of the lungs in the expiratory position, and 7 mm. for that exerted at the end of a normal inspiration. At the end of very deep inspiration the pressure may rise as high as 30 mm. (Donders).[13]

The amount of muscular tissue in the human lung is at most but scanty. Some authors, indeed, deny its existence, though in animals, such as the dog, it is certainly present, as proved by the contraction exhibited by the lungs of this animal when brought in contact with ice-cold water (Müller).[14] In the human subject, however, we may conclude that, so far as the lung proper is concerned, muscular action takes no direct part in the respiratory mechanism. The bronchioles, however, which are in intimate union with the texture of the lungs, are provided with unstriated muscular fibres innervated by motor nerves from the vagi. Stimulation of these nerves causes contraction of the bronchioles. Dr. Watson Williams[15] holds the view that the bronchioles normally expand and contract rhythmically, especially in children, the expiratory contraction being in comparative abeyance during quiet breathing in adults, and that in asthma the contractive phase of physiological respiration is merely exaggerated. He bases his view upon the acceptation by physiologists of afferent and efferent fibres to the bronchi in the pneumogastric nerve connected with the respiratory centre, and upon the fact that the larynx has rhythmic movements corresponding with those of respiration, which movements are, in very young children, shared by the alæ nasi.

Whilst the residual tension of the lungs has been thus carefully tested and measured, the corresponding thoracic tension *necessarily equal and opposite to it* has almost escaped observation; indeed, it has been deliberately misplaced and regarded as an expiratory force.

It was maintained by John Hutchinson,[16] Assistant Physician to the Brompton Hospital, in an able and elaborate paper read

before the Royal Medical and Chirurgical Society of London, that *inspiration is a wholly muscular act, the muscles in expanding the thorax having to contend against* (1) *the elastic resistance of the lungs;* (2) *the inertia and elastic resistance of the chest walls. Expiration, on the other hand, he believed to be effected by the elastic recoil of the lungs and chest walls;* and this theory of the mechanism of healthy breathing long found favour. The view cannot, however, be accepted without some modification. The recent work of Dr. Arthur Keith,[17] Professor Sherrington, and others, suggests that normal expiration should not be regarded as entirely the result of elastic recoil, but that the muscles of the abdominal wall regularly take some share in the performance of the act.

In regard to inspiration also the view embodies a fallacy. Thus it is incorrect to state that in quiet inspiration the muscles of the thorax have to contend against the inertia and elastic resistance of the chest walls, as well as that of the lungs. Far from this being the case, *the thoracic elasticity is a reserve force of appreciable power constantly tending to enlarge the chest, and acts, therefore, during quiet breathing, in favour of inspiration.* That this must be so would appear to follow, as a matter of reasoning, from what has been observed above respecting the state of tension in which the lungs are maintained, so long as they remain healthy, throughout life; but more fully to demonstrate the proposition, a brief reference may be made to some observations and experiments which bear upon the subject.

The late Dr. Salter[11] was the first to point out that at the commencement of breathing the elasticity of the chest walls should be placed as an inspiratory force. By a simple experiment he showed that in the dead subject, the thorax, when relieved from the traction of the lungs by an opening made into the pleura, expanded to the extent of $\frac{1}{100}$ inch. Paul Bert[18] (1870), in some ingenious experiments, recorded simultaneously the elastic pressure of the lungs and the expansion of the chest wall at the moment of opening the pleura of a recently killed dog. Traube,[19] in 1871, found that in the *living* dog the chest walls, on being relieved from the traction of the lungs, expanded in the manner indicated by Salter; but Traube does not seem to have been aware either of Salter's or of Bert's experiments, and he erroneously asserts that the

TABLE GIVING OBSERVATIONS ON THORACIC RESILIENCE IN TEN CASES POST-MORTEM.

(The observations were made with the co-operation of Dr. Coupland at the Middlesex Hospital.)

No.	Sex.	Age.	P.M.	Case.	Point of Application of Bulb.	Pleura opened.	Rise of Fluid in stem. Centim.	Rise of Fluid in stem. Total.	Equivalent Expansion of Chest. Millim.	Remarks.
1	M.	33	24 hrs.	Amputation	3rd L. cart.	*a*—2nd L. space *b*—2nd R. space	3·15 0	3·15	2·143	Right lung adherent, emphysematous; left free, slightly emphysematous; abdomen flaccid.
2	F.	38	21 hrs.	Tumour of jaw	3rd mid. st.	*a*—3rd space *b*—3rd space	.. ..	3·5	2·39	Slight further rise on making two fresh openings at fifth space; slight bronchitis.
3	M.	54	27 hrs.	Epithelioma of penis	4th mid. st.	*a*—3rd R. space *b*—2nd R. space *c*—3rd L. space	0 0 2·4	2·4	1·63	Right lung adherent, side more prominent; some emphysema of both lungs, mostly of the right.
4	F.	44	20 hrs.	Carcinoma of uterus	5th mid. st.	*a*—2nd R. space *b*—2nd L. space	2·3 2·3	4·6	3·19	Lungs quite healthy, no adhesions; collapsed to about one-fifth of bulk; rigor mortis, no decomposition; abdomen flaccid.
5	M.	27	23 hrs.	Delirium tremens	4th mid. st.	*a*—3rd R. space *b*—2nd L. space	0 0·3	0·3	0	Œdema of both lungs, recent pleural adhesions both sides.
6	F.	36	..	Comp. fract., bruised sternum	5th mid. st.	*a*—2nd R. space *b*—2nd L. space	0 0	0	0	Bronchitis; adhesions both sides.
7	M.	60	..	Fractured ribs, left side	4th mid. st.	*a*—2nd R. space *b*—2nd L. space	0 0	0	0	Cartilages ossified; right lung emphysematous; 3 pints of serum in left pleura.
8	M.	64	..	Strangulated hernia	4th mid. st.	*a*—2nd R. space *b*—2nd L. space	0·3 0	0·3	0	Lungs emphysematous and œdematous.
9	M.	41	12 hrs.	Fractured skull	4th mid. st.	*a*—2nd R. space *b*—2nd L. space	0 0	0	0	Lungs highly œdematous; dynamometer connected with trachea registered no pressure.
10	F.	56	40 hrs.	Carcinoma of uterus	5th mid. st.	*a*—3rd L. space *b*—3rd R. space	0 0·4	0·4	0	Rigor mortis passing off; abdomen distended; chest walls rigid, lungs emphysematous, but collapsed ½ inch.

General Remarks on Table.—The first four cases give positive results—viz., an actual expansion of chest on opening the pleural cavities=from 1·6 mm. to 3·19 mm. Case 4, giving the greatest expansion, was the only case in which the lungs were quite healthy. The last six cases give practically negative results—viz., no expansion or contraction in three cases, a small fractional expansion in one case, and a small fractional contraction in two cases. In all these latter six cases the lungs were notably affected by disease.

expansion, which he attributes to muscular action, is not obtained in the dead animal.

The appended table contains the details of ten post-mortem examinations made by one of us to estimate the extent to which the chest wall would expand of its own resilience when relieved from the traction of the lungs.[20a] The difficulty of finding the thoracic organs perfectly healthy in the dead subject is well known, and out of the ten cases operated upon, in only four instances were they approximately so.* The experiments were performed after the manner of Salter by taking a fine tube, A (Fig. 3), expanded below and having stretched across its expanded extremity, B, a piece of caoutchouc provided with a projecting button, C. The instrument, having been filled with coloured fluid, was so adjusted that the button was accurately applied to the third cartilage, the integuments having been previously reflected from the front of the chest. The level of the fluid having been noted on the Scale D attached to the stem, the thorax was cautiously opened, and an immediate rise of the coloured fluid was observed. A simple calculation enabled one to ascertain the actual expansion registered. The results of the experiments will be seen in the table; in the four comparatively healthy cases the expansions (forward movement) measured were 1·63 mm., 2·143 mm., 2·39 mm., and 3·19 mm. respectively. These figures, small though they are, correspond pretty well with the limits of expansion of the ribs during calm breathing. Thus Hutchinson calculated the costal movement in health at $\frac{1}{10}$ to $\frac{1}{4}$ line = 1 to 2 mm.; Sanderson gives 1·6 mm. Our own measurements, nineteen in number, recorded in the paper above referred to,[20b] by an instrument with writing lever,

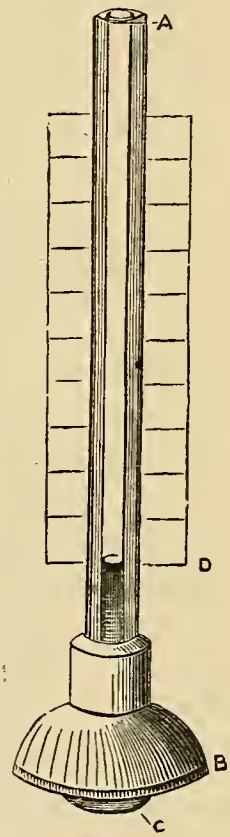


FIG. 3. — INSTRUMENT FOR ESTIMATING EXPANSION OF THE CHEST WALL.

* Professor Keith, in his reference to these experiments, omits to note that only in these four cases were the lungs approximately healthy, their action in the other cases being impaired by œdema, emphysema or pleuritic effusion.

would give rather a higher figure—viz., 2 to 3 mm. It would appear, then—and this is the point to which we wish especially to direct attention—*that in health, throughout ordinary inspiration, the limit of thoracic recoil, which we may call the reserve capacity of the thorax, is barely reached; and, therefore, that the sole resistance to be overcome by the inspiratory muscles is that of the lungs.*

This elastic *help* at the beginning and *hindrance* at the completion of the respiratory act is a spring-like function of the chest wall, the importance of which has not been duly recognised.

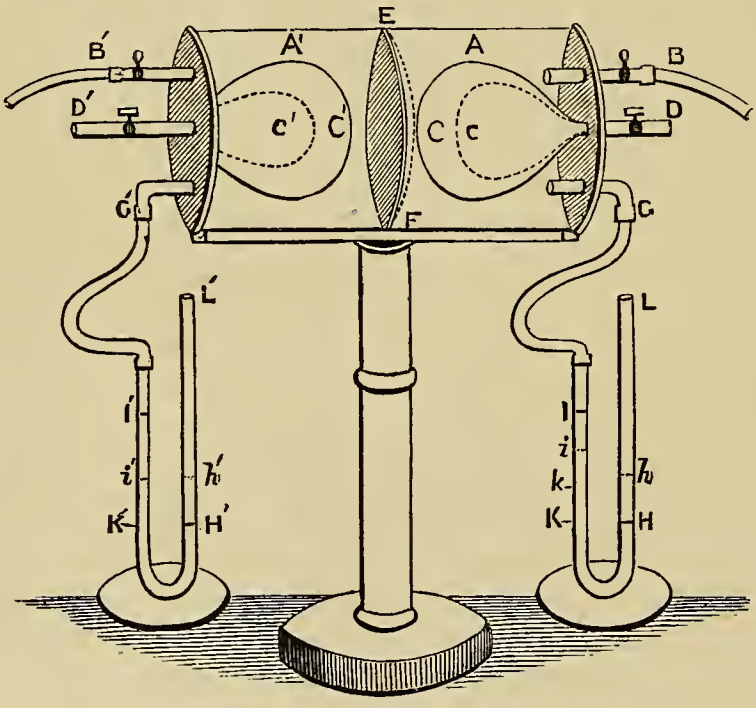


Fig. 4.—Diagram Model of the Chest.

In order more clearly to show the main physical conditions present in the chest, and how they are modified, (*a*) during normal respiratory movements, and (*b*) in certain diseases, the above schema or diagram-model of the chest was designed (Fig. 4).

The schema consists of a cylinder of glass, closed at each end by a metal plate screwed on and perforated for the admission of certain tubes. A central partition, EF, made of sheet india-rubber, divides the cylinder into two air-tight compartments, each of which, as will be presently seen, represents one-half of the thorax.

Tube D represents the trachea, and is connected with an elastic bag (*c*), representing the (left) lung. Tube G communicates with the space between the lung and the wall of the chamber, which space, therefore, corresponds to the pleural cavity. This tube is connected with a mercurial manometer, the free end of which, L, is open to the normal atmospheric pressure. Tube B also communicates with the same (pleural) space, and is provided with a stopcock and a mouthpiece.

Exactly the same parts are repeated on the opposite side of the partition, EF, which therefore represents the mediastinum.

The apparatus must be ascertained to be thoroughly airtight. Then, by partially exhausting the air from chamber A through tube B (trachea tubes D and D′ remaining open), we cause the bag *c* to expand to C, the mercury in the manometer, HKI, to rise towards the chamber, and the mediastinum, EF, to become convex, as indicated by the dotted line. By closing the stopcock B we maintain all the parts in this position.

If, next, we repeat the same process on the opposite side—partially exhausting chamber A′ through B′ until the mercury I′ is at the same level as I, the bag *c*′ will expand to C′, the mediastinum, EF, will again become vertical, and by closing stopcock B′, the parts will be maintained in this position of equilibrium on the two sides.

In the schema thus arranged, we have the conditions of the healthy chest rudely but accurately imitated. The two chambers represent the two sides of the chest, each containing a semi-expanded lung, C, C′, surrounded by a pleural cavity, A, A′ (here greatly exaggerated,* the cavity being rather potential than real in the healthy chest), each cavity being separated from that on the opposite side by the mediastinum EF, which is common to both and equipoised between them.

The walls of the natural thorax are, however, as we have seen, elastic or resilient in every part, although much more stiffly so than the lungs. We cannot exactly represent this resilience of the thoracic walls in our schema. The only parts

* This is unavoidable, since the walls of the schema are rigid, and if the bags fitted accurately, their further expansion in inspiration could not be represented. No fallacy is hereby introduced, however.

of our apparatus which are at liberty to yield to the excess of external atmospheric pressure over that within the pleuræ are the small surfaces of mercury at H, H′. Hence the elevation of the mercury, HI, H′I′, towards the chamber on each side, multiplied by the area of apertures G, G′, and divided by that of the whole surface of the chamber, would represent in millimetres the amount of recession of each portion of the thorax, provided each portion were equally resilient.

In considering this subject, we must not forget the diaphragm, which constitutes the floor of the chest, and which, owing to its resistance being weaker, yields far more than any other portion of the chest wall to the traction of the lungs, so that a recession of an inch on the part of the diaphragm is equivalent to the recession of only 1 or 2 mm. on the part of the ribs or cartilages. And yet it must be remembered that the diaphragm is not *per se* elastic, and that the limits of its actual *tension* are therefore very narrow. The elastic recoil of the diaphragm, in fact, depends mainly upon the spring of the cartilages to which it is attached. It is thus obvious that this muscular membrane must be drawn upwards and held in a state of tension by the lungs during the respiratory pause, their traction being in turn counterbalanced by the weight of the abdominal organs. Our mercurial columns, therefore, after all, very conveniently and fairly represent the whole thoracic resilience in a lump sum.

Having thus, with the aid of our schema, reviewed the statical conditions of the chest, the dynamics of respiration may be easily demonstrated.

In order to imitate an inspiration, aspiration must be made simultaneously through the tubes L and L′, thus representing the contraction of the diaphragm and inspiratory muscles on the two sides. This can be conveniently done by means of a syringe attached to a branched caoutchouc tube affixed to the extremities L, L′ of the manometers.

As the mercurial columns rise up the limbs L, L′ of the manometers, representing the expansion of the thorax, the lungs C, C′ enlarge by the entry of air through trachea tubes D and D′.

It will be observed that during the first part at least of inspiration the weight of the two columns of mercury, HI, H′I′, tells in favour of the inspiration. This weight of mer-

cury corresponds, as before said, to the outward resilience of the thoracic walls, and counterbalances the elastic traction of the lungs. This is a fact, well shown in the schema, and which has been already stated in the proposition, that *in health the resilience of the chest wall is in favour of inspiration.* Moreover, from the observations on the dead subject to which we have already referred, it would appear that *this elastic aid to inspiration obtains throughout the act in calm breathing.* Respiration is thus rendered smoother and less laborious, elasticity entering as an important item into the inspiratory, as it has been long known to do into the expiratory act.

The conditions present in the chest, as shown by the schema, must now be further examined in so far as they affect heart and circulation.

The disposition to the formation of a vacuum in the intrapleural space A, equal to the weight of the column of mercury HI, causes an aspiration towards that cavity which was at first shown by the convexity of the mediastinum EF (dotted line); and, there being a similar and equal aspiration towards the pleural cavity on the opposite side of the mediastinum, it follows that there is a constant determination of blood towards the cavities and walls of the heart—a hollow organ situated within the mediastinum, and communicating by a system of tubes with parts outside the thorax (see Fig. 5). This central attraction is forcibly overcome by the muscular contraction of the heart, but resumes its sway at the termination of systole, aiding the return of blood to the flaccid heart cavities, and encouraging the flow through the coronary vessels. This aspiration towards the heart, it must be remembered, is in health a constant force, increased during inspiration, held in momentary subjection during the more forcible muscular contraction of the heart, and not wholly extinguished even at the end of ordinary expiration.

In the subjoined drawing (Fig. 5), which depicts a somewhat more elaborate diagram model of the chest (but essentially corresponding with that illustrated by Fig. 4), the mediastinum is represented by a double layer of caoutchouc enclosing a space. With this is connected the manometer, D, which shows at a glance the negative pressure to which the heart is constantly subjected through the traction of the lungs from either side.

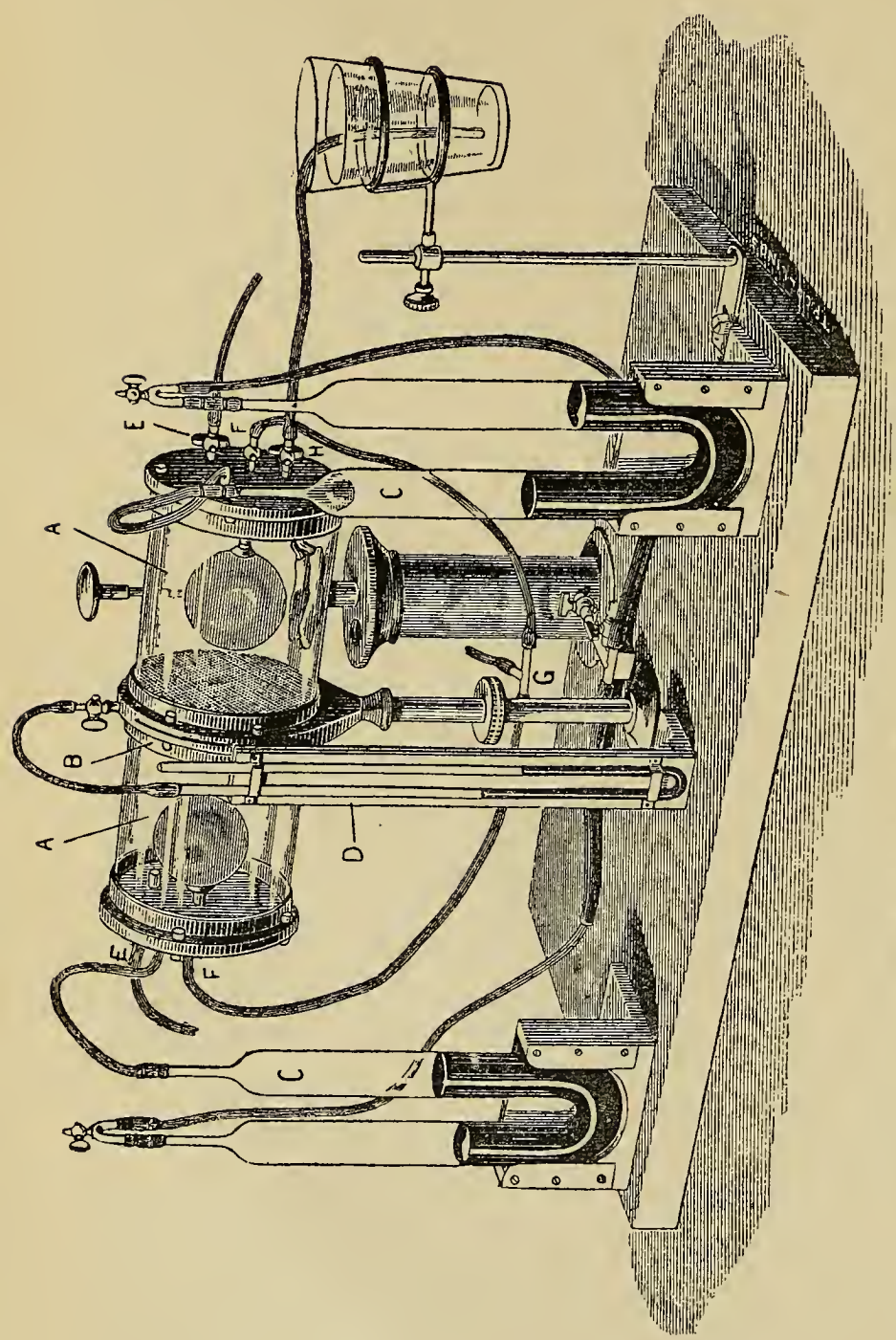


FIG. 5.—SOMEWHAT MORE ELABORATE DIAGRAM MODEL OF THE CHEST.

A, A, Lungs in position of moderate expiration within pleural cavities.
B, Mediastinal space (pericardium).
C, C, Manometers showing elastic traction of lungs upon chest walls.
D, Manometer showing negative pressure corresponding with traction of lungs within pericardium, as C, C show negative pressure within pleural sacs.
E, E, Tubes with stopcock (shut) communicating with pleuræ.
F, F, Bronchial tubes uniting in G, trachea.
H, Tube communicating with separate bag placed in pleural cavity A, for convenience in representing pleuritic effusions.

The instrument is represented as arranged by exhausting the air equally in the two chambers A and A, to show the condition of respiratory statics in the position of respiratory pause. This is effected either by exhausting the air equally in the two chambers A and A until the bladders representing the lungs become partially inflated, or (what amounts to the same thing) by inflating the two lungs through the trachea, whilst stopcocks E, E are open, then closing these cocks, and allowing the lungs to collapse to the point of equilibrium.

The reserve capacity of the chest to which we have referred, and which leads it, under certain circumstances and within certain limits, to enlarge without muscular effort from without or pressure from within, is a most valuable safeguard, and serves in some diseases to protect the lungs from pressure during temporary conditions of inflammation or engorgement.

If, for example, we look at a case of *pneumonia*, we find that, whereas on the healthy side the play of the chest wall is natural or increased, on the other side it is annulled. There is no retreat with expiration, and inspiration is checked by pain. On post-mortem inspection, we find the fine granulations of lymph evenly spread upon the pleural surfaces, showing no sign of pressure. The patient suffers little pain, except when he coughs or attempts to draw a deep breath. In fact, as the inflamed and consolidating lung increases in bulk, the thoracic wall, thereby released from its elastic traction, retreats to the position of inspiration; and this reserve capacity of the thorax is rarely exhausted in pneumonia, so that the swollen and tender lung as a rule escapes compression.

Similarly, in temporary *engorgement* and *œdema* of the lungs the consequences would be much more serious were it not for this reserve capacity of the thorax, which protects the organs from compression, save in extreme cases. In emphysema the lungs are rarely enlarged so as to be compressed by the ribs, the thoracic resilience being entirely neutralised only in very extreme cases.

With regard to *emphysema*, a diminution of the elastic spring of the chest wall is one of the first important features of the disease. In the normal chest, a deep breath is drawn by first making a deep expiration and then a deep inspiration. In emphysema the power of making a deep expiration is partially or entirely lost; the reserve capacity of the thorax is taken up, and it is in this direction that the trouble of emphysematous people lies. Regarding emphysema as in many cases essentially a disease of a degenerative kind, we must observe in the constant traction of the thorax upon the lungs a determining cause of their enlargement, as the chest gradually expands to the limits of its reserve capacity. If, on the other hand, as is often the case in early life, the

chest walls be soft and feeble, they cannot properly expand, and a small, narrow, or distorted chest results.

Lymphatics.—We have preferred to pass on from the consideration of the general structure of the lungs to a discussion of the mechanism of the respiratory function, and even, by way of illustration, to refer to some morbid states of perverted respiratory mechanism, before completing the anatomical description of the lungs by speaking of their lymphatic and nerve supply. This has been done with the view of bringing together those points of anatomy and function which are most concerned in the production of physical signs and symptoms.

In the lymphatic and nervous apparatus of the lungs we have, so to speak, the drainage and function-regulating systems, whose workings are for the most part hidden from us, to be brought to light mainly through the manifestations of disease. And, in truth, the important part played in pathology by the complex lymphatic system of the lung, as disclosed to us by the researches of the late Sir John Burdon-Sanderson[21] and Dr. Klein,[22] has become more clearly discerned with a better knowledge of the origin and extension of pulmonary diseases.

The lymphatics of the lung take their origin partly in a network of fine vessels which ramify in the connective tissue supporting the pulmonary lobules, partly in the mucous membrane of the bronchi. Those vessels which surround the lobules on the surface of the lung, communicate with the lymphatics of the visceral pleura, and both are drained by the *superficial* collecting trunks, which course over the surface of the organ and terminate in the glands at its root. The lymphatics, originating in the deeper lobules and in the bronchi, communicate with those on the surface of the organ, and are drained by the *deep* collecting trunks, which, under the name of "perivascular" and "peribronchial" lymphatics, accompany the pulmonary vessels and bronchi, to end also in the glands of the hilum.

The communication which exists between the superficial lymphatics of the lung and those of the visceral pleura shows how easily organisms may be carried to this membrane, and thus lead to its infection, even though recent research casts

some doubt upon the existence in the human subject of "stomata," whereby direct communication between the interior of the serous membrane and the lymphatics of the lung and pleura was believed to be established.

Another point of great interest insisted upon by Professors Poirier and Cunéo,[23] whose work on the lymphatic system has added much to our knowledge, is that the lymphatics of the pleural and peritoneal surfaces of the diaphragm communicate freely with each other, and that the one can be easily injected from the other. The frequent spread of infection between the two serous sacs is thus explained. Of great importance, too, is their observation that certain lymphatic vessels from the liver pass directly to, and drain into, the subpleural lymphatics of the diaphragm, thus explaining the frequent occurrence of pleurisy as a sequel to abscess and other infective diseases of the liver.

Nerves.—The nerve-supply to the lungs is derived from the anterior and posterior pulmonary plexuses, which are formed by the interlacement of branches from the vagi, joined by others from the sympathetic, chiefly from the inferior cervical ganglion, the annulus of Vieussens, and the ganglion stellatum. The nerves from these plexuses accompany and supply the bronchi and vessels, and then appear to end by forming a delicate network, the ultimate fibrils of which are distributed upon the alveoli.

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CHAPTER II

PHYSICAL EXAMINATION OF THE CHEST

Before proceeding to physical examination, preliminary inquiries have to be made into the health history of the patient, the circumstances which have led up to his present illness, and the chief symptoms which give him distress. In the course of these inquiries the appearance and manner of the patient may be noticed; and the experienced physician, skilled in the physiognomy of disease, will thus gain information of great value as a clue to the often confused story of the patient, and in suggesting to him further questions.

It would be folly to attempt to learn from a book the physiognomy of disease. Clinical study, an habitually careful scrutiny of the features and postures of actual sufferers, will alone enable us to appreciate it. The student will early distinguish the aspect of turgid lividity and laboured breathing of chronic bronchitis with dilated heart, from the pallor, anxiety, throbbing vessels, and dyspnœa with restlessness, of advanced aortic regurgitant disease. The hectic look of phthisis, the grave drawn lineaments of asthma, the puffy pallor of albuminuria, may in their more marked degrees be soon recognised even by the beginner. The finer traits and markings of disease, however, require more experience for their detection, and are of even more value in suggesting inquiries and often in leading one to suspect the presence of disease before sufficient signs can be found to justify a positive diagnosis. Incipient tuberculosis and obscure aneurism may be named as two instances in which the features of illness sometimes suggest a more guarded diagnosis than the physical signs would at first seem to warrant.

The relationship between physical signs and the diagnosis

of pulmonary disease is, again, of a strictly practical kind, although depending upon acoustic principles; and he who would become a successful auscultator and a good diagnostician must study auscultation in association with morbid anatomy. Thus will the stethoscope reveal to him at the bedside, in most cases, an accurate picture of the lung, heart or pleura under examination, as though the organ were exposed to his view. This intimate association in the mind between physical signs and the lesions which give rise to them is only to be acquired by combined clinical and post-mortem observation; no amount of reading or clinical work alone will suffice for its attainment.

It is the object of a textbook, however, to supply certain data for comparison, and to lay down principles and methods upon which a satisfactory exploration of the chest is best founded.

Shape of the Chest.—It is impossible in a word to describe the shape of the chest, but it may be said in the adult to be conical from above downwards, flattened in front, and grooved in the posterior median line, so that the antero-posterior diameter is about one-third less than the transverse. In the child these two diameters are more nearly equal. The shape of the upper portion of the chest is obscured by the pectoral muscles extending from the upper arm to the clavicle and ribs, giving it a somewhat square outline. In the female the apparent form of the thorax is still further altered by the mammary development.

Measurements of the Chest.—All departures from the normal, whether in shape, size or mobility, can be generally observed by inspection, but for accuracy in recording measurements are needed. For this purpose the *double tapes* and the *cyrtometer* are of value. The former instrument was introduced into clinical use by the late Dr. Charles J. Hare, Physician to the University College Hospital, and consists of two tapes connected by a central piece for adaptation to the spine, from which point each tape is graduated. The back piece being carefully held over the spinous processes, and a mark being made exactly in the median line of the sternum, the two tapes are brought round the chest at the same level and with moderate firmness of application, to overlap one another at the front line. A comparative measurement of

the two sides is thus at once read off, the sum of the two measurements giving the total circumference.

By holding the tapes lightly the expansion of the chest can be measured during calm breathing, and by making the patient take a deep breath, followed by a full expiration, the total expansibility is ascertained; also, what is equally of value, the contractility of the chest beyond the point of ordinary expiration. Finally, a comparison in all these respects can be made between the two sides.

By means of the *cyrtometer*, a tracing of the circumferential outline of the chest is obtained. This instrument, in the form of jointed whalebone, was first employed by Woillez.[1] Its most convenient form is that suggested by Dr. Gee,[2] and consists of two pieces of thin lead piping connected by a hinge of rubber tubing. The hinge is carefully applied over the spinous process at the level required, and the piping moulded round the chest until the two ends cross one another in the median line in front; a mark is then made and the instrument allowed to fall away from the chest, being held by the flexible joint. It is subsequently adjusted in position on a sheet of paper, and "sternum" and "spine" being marked, the pencil is carried round the inner circumference, and an exact outline of the shape of the chest is thus obtained, any comparative or general alteration being readily observed.

Callipers are sometimes used for taking comparative antero-posterior measurements, but, owing to many practical difficulties, they are not of much value for clinical purposes.

The circumference of the chest varies considerably in different individuals and within the range of health. Thus, Walshe[3] noted in adult males of medium height measurements ranging between 27 and 44 inches in the circumference taken opposite the sixth rib. Such extreme measurements are, however, exceptional. The admirable statistics of Baxter,[4] dealing, without selection, with over 300,000 of the adult male population of the United States, between the ages of eighteen and forty-five, at the time of the American Civil War, show the average circumference of the chest to be between 33 and 34 inches (see table, p. 27). For clinical purposes these absolute measurements do not teach us much. Relative measurements of the two sides are, however, of value in certain diseases attended with enlargement or dimi-

nution of the chest on one side, and in such cases cyrtometer tracings are helpful in giving exact information also as to shape.

The mobility of the chest is of more importance than its mere size, as it affords a better indication of vital capacity. In the healthy adult the difference between extreme inspiration and extreme expiration should not be less than $2\frac{1}{2}$ inches, as measured by the tapes at or about the level of the nipples. It may amount to as much as 5 inches. The difference should be nearly equally divided between the two sides, a slight excess in favour of the right being of no importance. Vierordt,[5] indeed, states that in right-handed persons there is constantly an excess of from 1 to $1\frac{1}{2}$ centimetres (about $\frac{1}{2}$ inch) on the right side.

In calm breathing, however, the actual movement of the chest is very small, averaging in the healthy male (according to Walshe) $\frac{1}{4}$ inch: the expansion of any one spot of the chest surface not exceeding 2 to 3 millimetres, as already shown (p. 12). The whole subject of mensuration will be found duly discussed in Walshe's treatise, to which we have already referred. For detailed researches respecting the movements of individual ribs, we must refer the reader to Dr. Ransome's[6] monograph on stethometry, and Dr. Arthur Keith's[7] more recent work on the subject.

Pneumatometry.—The power ordinarily employed by the inspiratory and expiratory forces during calm breathing, and that which they are capable of exercising during extremest effort, have been carefully estimated by various authors, and especially by Waldenburg.[8] Pneumatometry and Spirometry are most fully discussed in his work, and later researches have added but little to our knowledge of the subject.

The instrument employed by Waldenburg consisted of a manometer provided with a naso-oral mask so padded as to fit with accuracy, each limb of the manometer measuring about 12 inches (270 millimetres), and being half filled with mercury.

For calm breathing the mercurial surface would indicate a difference of from 1 to 2 millimetres. With forced inspiratory effort the mercury could be maintained at a minus pressure of $2\frac{1}{2}$ inches (60 millimetres), nearly double this pressure being momentarily obtainable. The expiratory force ex-

ceeded the inspiratory by 20 to 30 millimetres—*i.e.*, with forced expiratory effort 3½ inches (90 millimetres) of positive mercurial pressure might be maintained, and, momentarily, as much again.

Marked variations from the healthy standard are met with in disease, and in two directions:

1. *The inspiratory power is diminished whilst the expiratory (except in extreme cases) remains normal.* This type is observed in phthisis, even in the earliest stages of that disease. It is found also in laryngeal, tracheal and bronchial obstructions, and to a less extent in pneumonia or in pleuritic effusion.

2. *The expiratory pressure is lowered, the inspiratory remaining normal or being even increased, or subnormal, but in all cases remaining relatively higher than the expiratory.* This type obtains in emphysema, bronchitis and asthma, also in diseases of the abdominal organs which impede the play of the expiratory muscles.

Spirometry.—In the healthy adult at rest the respirations number from sixteen to twenty per minute; they are somewhat slower during sleep than when awake, and are readily accelerated by movements, effort, or excitement of any kind. With each act of calm breathing there is an influx into the chest, followed by a corresponding efflux, of some 500 c.c. (30 cubic inches) of *tidal air.* If the inspiration be an unusually deep one, an additional 1,500 c.c. of so-called *complemental air* may be inhaled; while, conversely, after a normal expiration 1,500 c.c. of *reserve* or *supplemental air* may be still further expelled, if a very forcible expiratory effort be made. The sum of these three, representing the amount of air which can be expelled by the deepest expiration following the deepest inspiration, constitutes the *vital capacity of the individual,* and in a healthy Englishman of average build amounts to rather more than 3,500 c.c. (210 cubic inches). Even then, however, the lungs are by no means airless, some 1,000 c.c. of *residual air* being still retained within them, which can never be expelled during life.

These facts were originally worked out by Hutchinson,[9a] by means of an instrument called the spirometer, which consisted essentially of a graduated gasometer nicely balanced and provided with a mouthpiece, through which the patient could

breathe into the meter previously set at zero. By a series of elaborate investigations, he arrived at the following important conclusions, which have not been altered or materially added to since his original paper.

Hutchinson found that the vital capacity varied with the *height* in a very definite manner, as will be seen by the subjoined table:

Height.	Vital Capacity. From Observation.	Hutchinson. From Calculation.	Weight.	Circumference of Chest. (Number of Cases examined in Brackets) Baxter.
ft. in. ft. in.	cub. in.	cub. in.	lb.	in.
5 0 to 5 1	174	174	120	30·8 (1,674)
5 1 ,, 5 2	177	182	126	31·1 (9,871)
5 2 ,, 5 3	189	190	133	
5 3 ,, 5 4	193	198	136	31·9 (36,989)
5 4 ,, 5 5	201	206	142	
5 5 ,, 5 6	214	214	145	32·9 (76,157)
5 6 ,, 5 7	228	222	148	
5 7 ,, 5 8	229	230	155	33·6 (94,450)
5 8 ,, 5 9	237	238	162	
5 9 ,, 5 10	246	246	169	34·2 (64,591)
5 10 ,, 5 11	247	254	174	
5 11 ,, 6 0	259	262	178	34·7 (25,500)

In the table the two columns of vital capacity—one taken from a number of observations, the other from calculation—are so nearly identical that we may take it that every inch in stature above 5 feet should add 8 cubic inches to the vital capacity. The last column has been added to show the corresponding circumferential measurements of the chest.

Hutchinson[9b] further showed that *body-weight* influences the vital capacity. At the height of 5 feet 6 inches the vital capacity increases in the ratio of 1 cubic inch per pound, as the body-weight rises from 105 pounds to 161 pounds (11½ stone). Above this limit, however (up to 196 pounds, or 14 stone), as the person becomes heavier the capacity diminishes in the same proportion, losing 1 cubic inch per pound as the weight increases.

After thirty and up to sixty years of age, there is a decrease of nearly 1½ cubic inches per year of age. In disease the vital capacity decreases from 10 to 70 per cent. This diminution is dependent upon, but not directly proportional to, the extent of breathing surface encroached upon, since the lung or por-

tion of lung remaining healthy may take a compensatory action (Waldenburg).

Topography of the Chest.—For convenience in clinical examination and description, the chest is mapped out into cer-

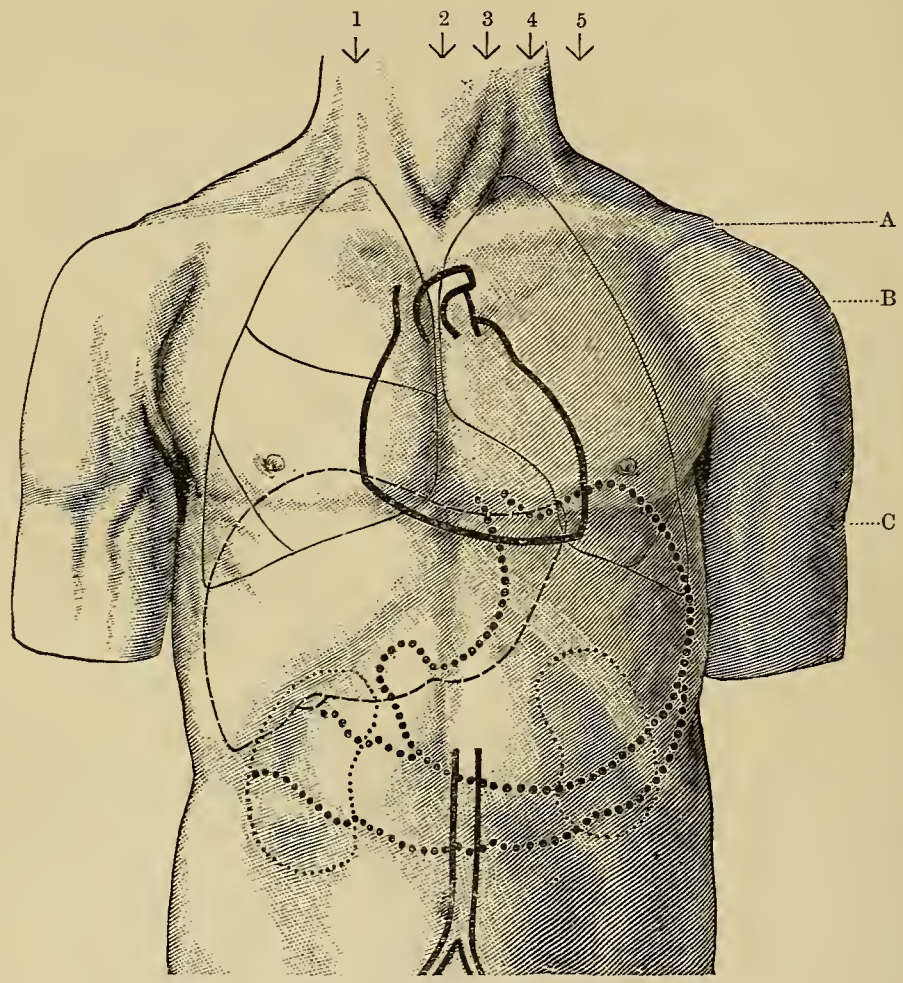

FIG. 6 (FROM QUAIN'S "ANATOMY").

A, Upper margin of sternum = intervertebral disc between second and third dorsal vertebræ; B, second costal cartilage = fifth dorsal vertebra; C, infrasternal depression (xiphisternal articulation = intervertebral disc) between ninth and tenth dorsal vertebræ (spine of the eighth dorsal vertebra). The lines of longitudinal parallels are indicated by figures and arrows above the diagram.

N.B.—The line of the secondary fissure should run more horizontally across the chest at about the level of the fourth rib.

tain regions. These are sufficiently indicated by the terms employed, viz.:

Anteriorly, the supraclavicular, clavicular, infraclavicular, mamary and inframammary regions, on the right and left sides respectively.

In the median line, the suprasternal, upper sternal and lower sternal regions.

Laterally, the axillary and infra-axillary regions.

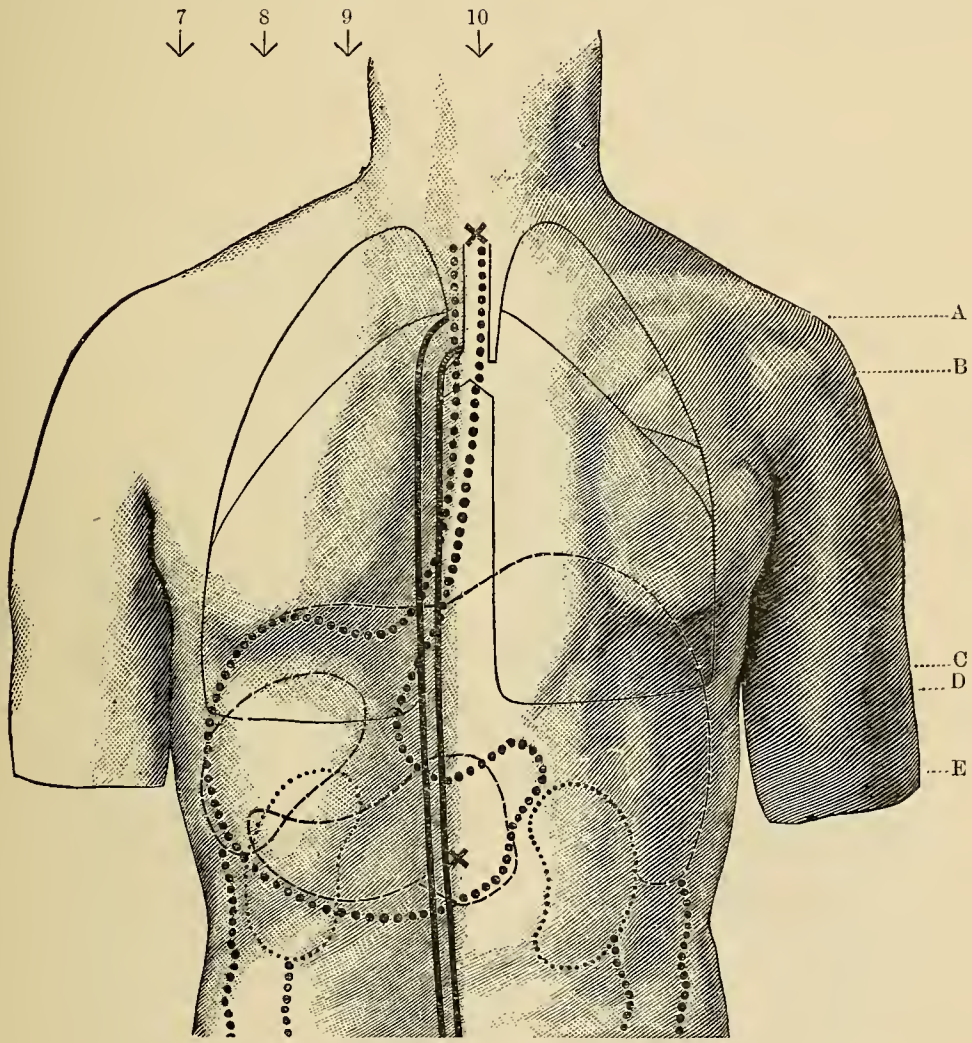

FIG 7 (FROM QUAIN'S "ANATOMY").

X, Seventh cervical spine = apex of lung; A, commencement of great fissure (apex of lower lobe) = tip of spine of second dorsal vertebra—*i.e.*, 2 inches below summit of lung; B, bifurcation of trachea = lower part of body of fourth dorsal vertebra, or between the third and fourth dorsal spines (level of junction of manubrium and gladiolus sterni anteriorly); C, long axis of spleen = tenth rib; D, base of lungs = tenth dorsal spine; E, upper end of left kidney = eleventh dorsal spine, the right being ½ inch lower; X = first lumbar spine. The lines of longitudinal parallels are indicated by figures and arrows above the diagram.

Posteriorly, the upper scapular (supraspinous), the lower scapular (infraspinous), the interscapular and the basic regions on each side.

For the purpose of more accurately noting for future reference the locality of any particular physical signs, there is no better or simpler plan than that of employing imaginary lines and levels drawn upon the chest surface (see Figs. 6 and 7). Thus the chest can be mapped out in latitude and longitude

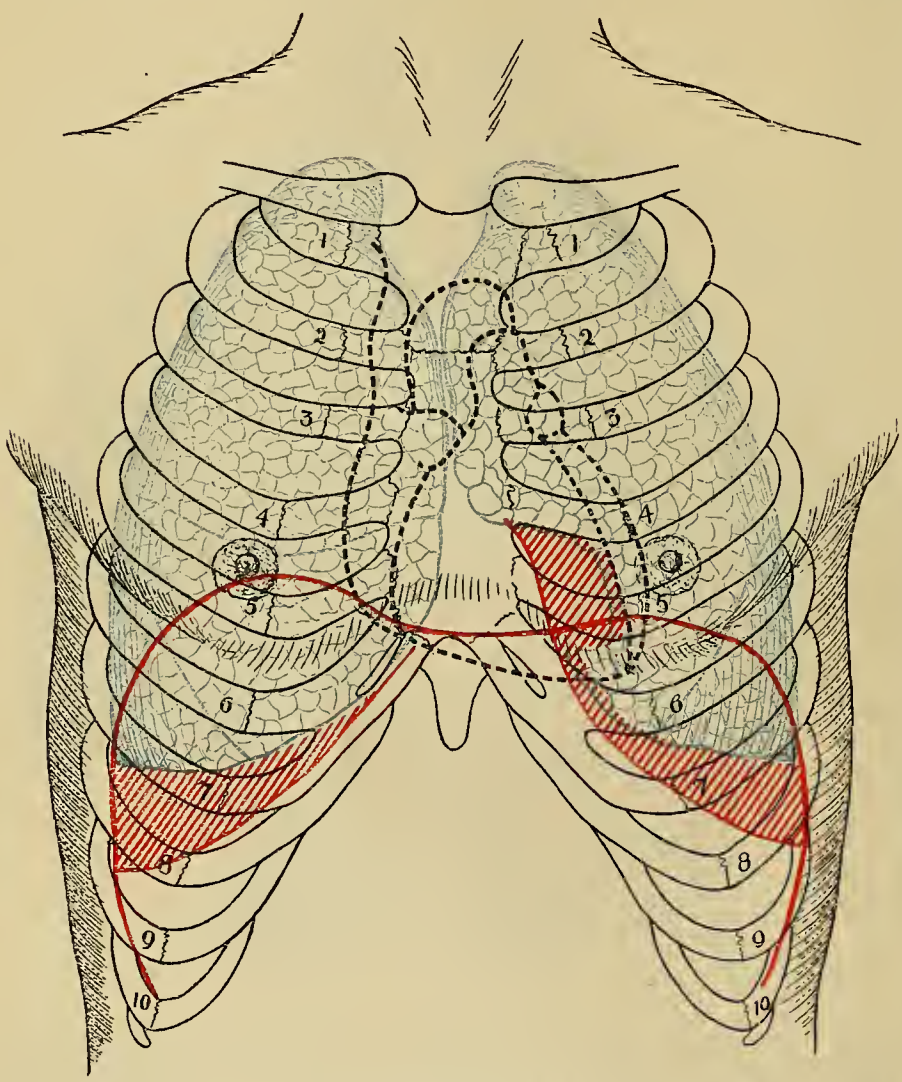

FIG. 8.—SHOWING THE RELATION OF THE THORACIC VISCERA TO THE CHEST WALL.

(After Professors Merkel and Thane, slightly modified.)

by parallel *vertical* lines drawn from summit to base through the *mid-sternal* (1), *parasternal* (2), *sterno-nipple* (3), *nipple* (4), *anterior axillary* (5), *mid-axillary* (6), *posterior axillary* (7), *mid-scapular* (8), *interscapular* (9), *and vertebral lines* (10), intersected by parallel lines drawn *horizontally* at the levels of the several rib cartilages in front and the several spinous pro-

cesses behind, with the addition of *nipple level, ensiform level,* etc.

A careful observation of the accompanying diagrams (Figs. 6 and 7) will impress upon the memory the main features in

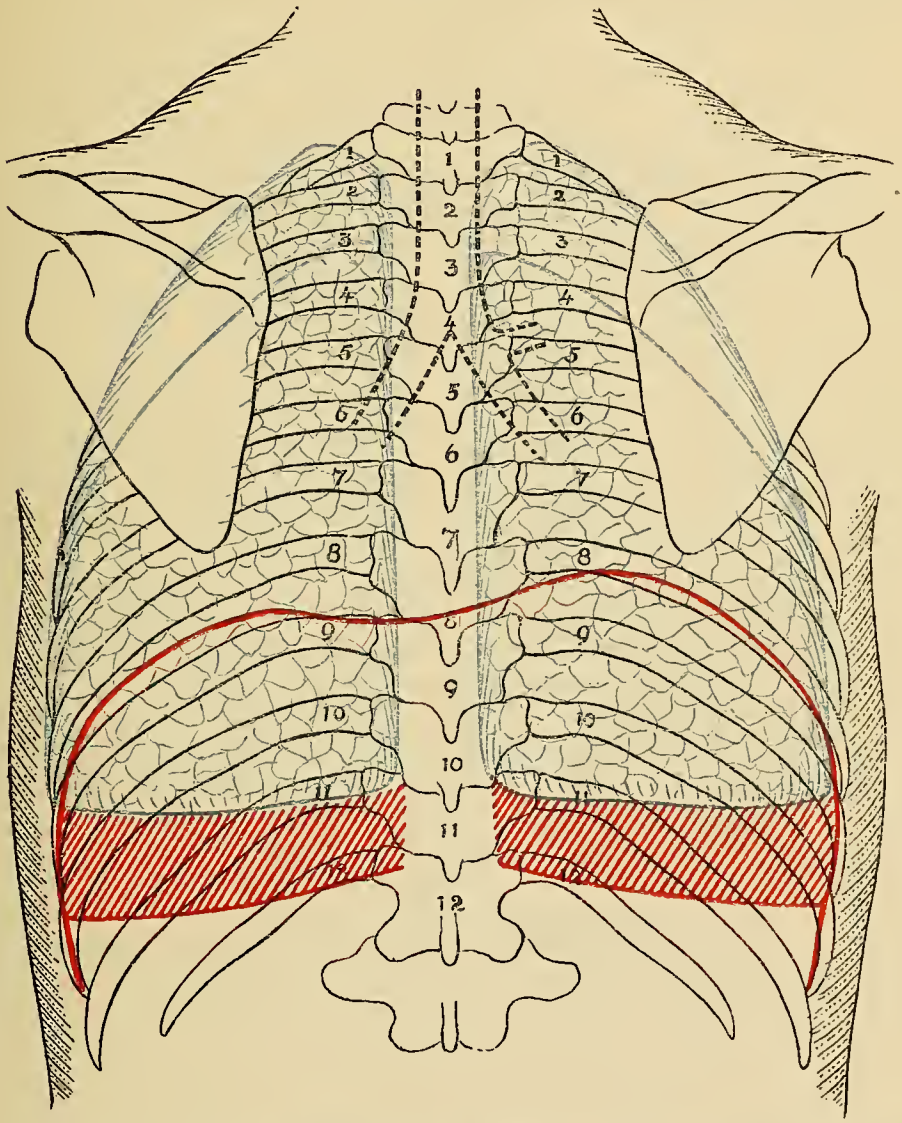

FIG. 9.—SHOWING THE RELATION OF THE THORACIC VISCERA TO THE CHEST WALL.

(After Professors Merkel and Thane, slightly modified.)

the topography of the chest organs and of those of the abdominal viscera in immediate relation with the chest. Marginal references are made at certain levels to facts of topography, which it is of some importance to bear in mind in clinical work. It will be observed that the upper or anterior

lobes of the lungs occupy most of the front aspect of the chest, whereas posteriorly the lower or posterior lobes correspond with nearly the whole surface. The relations of the thoracic viscera to the chest wall are shown more in detail in Figs. 8 and 9.

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[7] "The Mechanism of Respiration in Man," by Dr. Arthur Keith, with bibliography, in *Further Advances in Physiology*, edited by Leonard Hill, M.B., F.R.S., p. 182. London, 1909.

[8] *Die pneumatische Behandlung der Respirations- und Circulationskrankheiten*, von Dr. Med. L. Waldenburg. Berlin, 1880.

[9] (*a*) "On the Capacity of the Lungs and on the Respiratory Functions," by John Hutchinson, *Transactions of the Royal Medical and Chirurgical Society of London*, 1846, vol. xxix., p. 137.
(*b*) *Loc. cit.*, pp. 164 and 174.

CHAPTER III

PHYSICAL EXAMINATION OF THE CHEST (*Continued*)

In physically exploring the chest, *inspection*, *palpation*, *percussion* and *auscultation* are successively employed.

Inspection.

The chest should always, if possible, be uncovered, so that a general view of its conformation can be obtained. We may thus observe the broad, well-formed chest of robust health, or the small, narrow, long chest adapted to small lungs, with antero-posterior and lateral diameters diminished, costal angle* narrowed, and ribs unduly oblique and approximated; or, again, the thorax may be expanded, with widened intercostal spaces, straightened ribs, increased costal angle, and deepened antero-posterior diameter—making up the round-shouldered, barrel-shaped chest suited to the accommodation of enlarged lungs. Further, the thorax may be distorted by various kinds of spinal curvature, by rickets in early life, or by continued pressure in any particular direction. Finally, there may be local flattenings or bulgings of the chest walls, due to alterations in the subjacent viscera, and giving rise to a want of symmetry on the two sides.

The *movements* of the chest are of great importance in diagnosis. The free and equable expansion of the chest implies the free entry of air into the lungs; on the other hand, relative immobility or recession of any portion of the chest during inspiration signifies that the entry of air to the corresponding portion of lung is, from some cause, retarded or impeded. In cases of general obstruction to the entry of air, whether by impediment at the main air-passage or through-

* The angle formed by the inferior margins of the chest at the ensiform cartilage. This angle should measure nearly 90 degrees.

out the bronchial tract, there is universal recession of all the soft parts during inspiration—the supraclavicular regions sink downwards, the hypochondria recede, and the intercostal spaces deepen during the effort to expand the chest against atmospheric pressure. When the difficulty of expansion, whether from intrinsic disease of the lung or pleura, or from obstruction of air-passages, is restricted to one side of the chest or to a portion of one lung, the restrained expansion during inspiration is limited to that portion. Thus from inspection alone we may often form an opinion as to the seat, and even surmise the nature, of the disease present.

Various instruments which have already been referred to—callipers, cyrtometer, double tapes—are valuable for the purpose of *recording* differences in shape and measurement, but the information which they are useful in recording is at once obtained by the eye of the trained observer.

By inspection we thus learn: (1) whether a patient be large-chested or small-chested; (2) whether the shape of the chest be good and symmetrical, deformed, flattened, or bulged in any of its parts; (3) whether its movements be free and equable, or irregular and restricted, generally or locally, and whether increased or diminished in frequency. Lastly, any surface markings, such as enlarged veins, tumours, or abnormal pulsations, will at once attract the eye and be duly noted.

Palpation.

Palpation is employed in aid both of inspection and percussion.

(*a*) During preliminary inspection of the chest the position of the heart's apex-beat should invariably, and as a matter of habit, be ascertained, and any deviation from its normal position—viz., the fifth intercostal space half an inch to the sternal side of the left nipple line—should be noted.

(*b*) Any local bulging or tumour will naturally be manipulated to ascertain its relation with bone or soft structure, whether it be solid, fluctuating, or pulsating; also any tenderness will be observed.

(*c*) The expansion, symmetrical or otherwise, of the two sides will be observed by placing the hands evenly on the two sides of the chest.

(*d*) In connection with percussion the observer should notice differences of resistance as well as of tone.

(*e*) Increase or diminution of *vocal vibration* or *fremitus* will be noted over any spot of altered resonance by applying the hand, and making the patient utter some resonant words, such as "ninety-nine."

Vocal fremitus is increased by consolidation of lung, provided the bronchi be not occluded; diminished by much thickening of the pleura, by obstruction of the main bronchus, or by air in the pleura; annulled by fluid in the pleura, if in sufficient quantity. The loudness or feebleness of the voice, as well as height or depth of pitch, must of course be taken into account in judging of fremitus, and corresponding parts on the two sides should always be compared. Any vibration from above can be checked by applying one hand lightly and edgewise to the chest above the part which is being palpated.

(*f*) Loud, coarse, bronchial râles may cause the chest walls to vibrate perceptibly, producing *rhonchal fremitus;* sometimes cavernous or large crackling râles will do the same. Pleuritic friction may likewise be perceptible to the hand—*friction fremitus.* In cases of effusion into the pleural cavity, or in hydatid cysts near the surface, *fluctuation* may sometimes be elicited.

Percussion.

Percussion is the method by which we test the resonance of various parts of the thorax, and compare it with that which experience has found to obtain in health. The varying degrees of resonance depend upon the relative amount of air and solid structure subjacent, and upon other causes to be incidentally noticed.

It is interesting to note that while inspection and palpation have been practised since the earliest times, percussion, as a means of diagnosis in diseases of the chest, is of comparatively recent origin. It was first employed by Auenbrugger,[1] a Physician of Vienna, in 1761, but its importance was misunderstood, and it was not until 1808, ten years before the discovery of auscultation by Laennec, that Corvisart, of Paris, Laennec's teacher, brought Auenbrugger's forgotten work to notice, and the method came into general use.[2a]

Method of Percussing.—It is best to use the fingers only for percussing. One finger of the left hand should be placed firmly upon the chest so that the two last phalanges are accurately applied to the part percussed. With one or two of the fingers of the right hand, semi-flexed, the percussion should be made, so that the stroke fall vertically upon the applied or *pleximeter* finger. Be it observed:—

1. That the pleximeter finger be applied accurately and with sufficient firmness.

2. That it be applied precisely in the same manner and to the same spot on the corresponding sides of the chest in comparing them; for instance, the finger must not be applied along the intercostal space on one side, and across the ribs on the other.

3. The percussion stroke must be made from the wrist, quite vertical to the surface percussed, and in comparing two spots the force of percussion must be the same. These conditions cannot be nearly so well preserved if percussion be made from the elbow.

4. As a rule, the percussion stroke should be light. The precise limits of dulness, whether in chest or abdomen, cannot be ascertained by hard percussion, since the vibrations of collateral parts are too strongly elicited. It is laid down in most textbooks that percussion should be made "staccato," the percussion finger not being allowed to rest upon the pleximeter. This, we are convinced, is an error, which is avoided by non-observance by nine out of ten of the best manipulators in actual practice. It is sometimes, of course, advisable to employ the lightest possible staccato percussion, and it is also sometimes necessary to employ hard percussion to elicit dulness or resonance of deep-seated parts.

5. The sense of touch must be used equally with that of hearing in percussion, and the degrees of resistance appreciated by the pleximeter finger are to be carefully noted. In this way the hardness and want of resilience over dense subjacent tissues may be readily *felt*.

Various pleximeters and plessors are used by some observers, modifications of those originally designed by Piorry.[3] The simplest pleximeter consists of a piece of ivory some two inches long and half an inch broad, the plessor being a small hammer with an india-rubber tip to the striking

surface. These instruments may be of value in demonstrating to a class, but their employment by students should not be encouraged for three reasons:

1. We may rely upon having our fingers with us, but are apt to leave detached instruments behind.

2. Patients when very ill are frightened or annoyed by instruments, and may be readily hurt by them.

3. The important reason is, however, that in using such instruments we deprive ourselves altogether of the information gained by the sense of resistance. Some physicians are inclined of late to take to the pleximeter, still using the fingers as the plessor; but inasmuch as it is the pleximeter finger with which we chiefly appreciate resistance, the objection in greatest measure still holds good.

The position of the patient during physical examination of the chest by percussion and auscultation is of importance. If not in bed, the sitting posture, with the back supported by a cushioned chair, is the best for examining the front of the chest; if in bed, the semi-reclining posture with the back firmly supported. Whilst the back is being examined the patient should be directed to lean slightly forward, and to let the arms fall loosely down between the knees, in which position the suprascapular regions, at which the summits of the lungs are situated, are best exposed. If the patient stoops forward too much with folded arms and bent back, the respiratory movements are impeded, and a very considerable amount of dulness may be developed at the right base by the thrusting backwards of the upper posterior surface of the liver.

Each region of the chest surface should be systematically tested by percussion, the two sides of the chest being at all points compared. Not only, however, must the two sides of the chest be thus compared from above downwards, but percussion should be employed from side to side across the sternum, so as to define the limits of the anterior margins of the lungs from either side. By this means valuable information is often elicited in cases of consumption, new growths, and pleuritic effusions. It is useful in practice, having ascertained the lower limit of resonance, to direct the patient to make a full inspiration and expiration whilst repeated light percussions are being made. The pleximeter finger can follow up and down the level of resonance, and in this way

valuable information as to the mobility of the diaphragm and expansibility of the bases of the lung can be obtained.

In the healthy chest we obtain during moderate expiration *superficial cardiac dulness.* This corresponds roughly with a triangular area, marked off by a line curving outwards towards the apex, from the lower margin of the fourth left costal cartilage at its junction with the sternum, by a second line running vertically down the centre of the sternum, whilst the base is formed by a horizontal line passing from the apex-beat to the mid-sternum. On firm percussion the *deep cardiac dulness* may be elicited as high as the third left cartilage and the right margin of the sternum. *Stomach note* is obtained at the sixth costal cartilage in the left mid-sterno-nipple line; and in the left postero-axillary line a small area of dulness extending from the ninth to the eleventh rib and reaching to within two inches of the middle line posteriorly, marks the situation of the *spleen.* On the right front of the chest, in the nipple line, *liver dulness* is elicited below the level of the sixth rib, and on deep percussion, for a rib higher; posteriorly about two fingers' breadth of dulness at the extreme right base marks that portion of the liver which is here in contact with the chest wall. Lung resonance is obtained anteriorly as high as the supraclavicular fossa, and posteriorly extends upwards to the level of the seventh cervical spine, *i.e.*, about one and a half inches above the level of the clavicle. It is in these higher regions of the chest, corresponding with the apices of the lungs, that the first evidence of tuberculous disease is usually detected.

In disease the percussion note is altered in various ways, as may be seen from the following table, which includes all the terms used in a technical sense, which are necessary for describing the sounds met with in diseases of the chest. They are—with one or two unimportant additions—those which were carefully selected by the late Dr. Mahomed and by Dr. Douglas Powell, acting as English members of a committee nominated at the International Medical Congress held in London in 1881, to endeavour to simplify the terminology of auscultation for international use.

Terms.	Definition.	Synonyms.	Common Significance.
PALPATION.			
Vocal fremitus. Normal.	The transmission of laryngeal vibrations to the chest wall, appreciable by the hand.		
Increased.	. . . . .	. . . . .	Consolidation of lung.
Diminished.	. . . . .	. . . . .	Bronchial obstruction, or separation of lung from parietes by thickened pleura.
Absent.	. . . . .	. . . . .	Effusion of fluid or air in the pleural cavity.
Rhonchal fremitus.	Transmission of the vibration of rhonchus to the hand applied to the chest.	. . . . .	Partial obstruction of larger bronchi. Bronchitis.
Friction fremitus.	Transmission of the vibrations of pleuritic friction.	. . . . .	Pleuritic roughening.
PERCUSSION.			
Normal resonance.	An arbitrary term signifying the varying degrees of resonance of the different parts of the chest within the range of health.	. . . . .	Health :—needs confirmation by other signs.
Increased resonance.	. . . . .	Hyper-resonance.	
Tympanitic resonance.	Drum-like note.	. . . . .	Air in the pleural cavity.
Skodaic resonance.	A peculiar form of tympanitic resonance of high pitch and great clearness.	Relaxed lung note.	Lung in contact with chest wall relaxed, but not compressed, by moderate effusion into the pleura. Central consolidation in pneumonia will sometimes produce this note.
Impaired resonance.	. . . . .	Dulness of different degrees. Hardness. Wooden percussion.	Incomplete consolidation ; coexisting *increased resistance* may often be appreciated during percussion.
Absence of resonance.	. . . . .	Absolute dulness. Tonelessness. Flatness.	Consolidation of lung, or its displacement by fluid or tumour.
Amphoric resonance.	The modified impairment of resonance sometimes elicited over a cavity, and often accompanied by "cracked-pot sound" (*bruit de pot fêlé*).	Tubular note.	Pulmonary excavation near the surface and freely communicating with the bronchi. Normally obtained on percussing the trachea with the glottis open.

Terms.	Definition.	Synonyms.	Common Significance.
AUSCULTATION. Breath-sounds. Vesicular.	Inspiratory sound soft and breezy; expiratory sound shorter, weaker, or even absent. There should be no perceptible pause between the inspiratory and expiratory sounds.	Normal breath-sounds.	Healthy lung.
Exaggerated.	Intensified normal breath-sound, due to increased movement of tidal air.	Puerile. Compensatory. Supplementary.	Increased function. When heard over a portion of one lung signifies compensatory action to make up for deficiency or disease elsewhere. Normal in young children, and in adults during violent exercise of the lungs.
Weak.	Deficient movement of tidal air.	Feeble. Partial suppression.	Diminished function.
Suppressed.	. . . . .	Absence of breath-sounds.	Lung distant from surface, or bronchus obstructed.
Interrupted.	Inspiratory sound partially or completely divided into two or three sounds.	Jerking. Wavy. Cog-wheeled. *Respiration saccadée.*	Irregular expansion of lung from partial consolidations about small bronchi; but often of purely nervous origin through irregular contraction of muscles.
Prolonged expiration.	Expiration lengthened to or beyond duration of inspiration.	. . . . .	Partial consolidation of lung or partial obstruction of bronchi.
Broncho-vesicular.	The vesicular part of the breath-sound being partially annulled, the tubular or glottic portion of that sound is heard with greater distinctness, especially during expiration which is prolonged.	Harsh. Coarse. Sub-tubular.	Commencing consolidation. Some authors use the term as descriptive of the roughened breath-sound of dry catarrh of the larger bronchi. (Heard normally in neighbourhood of bronchi.)
Bronchial.	A blowing breath-sound, the inspiration and expiration being about equal in pitch and duration, and distinctly divided. Placing mouth in position to pronounce word commencing with guttural *ch* (χ), and drawing breath to and fro, imitates the sound with exactness (Skoda).	Tubular. Blowing. Tracheal. May be high-pitched or whiffing, medium, or low-pitched.	Hepatisation or condensation of the lung. (Heard typically over trachea.)

Terms.	Definition.	Synonyms.	Common Significance.
Breath-sounds (*continued*).			
Cavernous.	A blowing breath-sound of hollow quality, most so in the expiratory portion, which is usually of lower pitch than the inspiratory.	. . . . .	Pulmonary excavation, or condensed lung with dilated bronchus.
Amphoric.	Similar to the above, but with blowing character and hollowness intensified.	. . . . .	Large pulmonary cavity; sometimes heard in pneumothorax.
Adventitious sounds. Râles.	Adventitious sounds produced in the bronchi and lungs under diseased conditions by the passage backwards and forwards of the tidal air.		
Varieties— 1. *Dry râles.* Sonorous râle.	A low-pitched, loud, snoring sound, produced by partial obstruction in a large bronchial tube, which imparts vibrations to the air-current.	Rhonchus.	Bronchitis of the larger air-tubes.
Sibilant râle.	A high-pitched, whistling sound, produced in the same manner as the rhonchus, but in a smaller tube.	Sibilus.	Bronchitis, or spasmodic narrowing of the medium or fine tubes.
Sonoro-sibilant râle.	The commingling of the two former sounds.		
Stridor.	A coarse, vibrating rhonchus, generated at the larynx, or by pressure on the trachea or main bronchus.	Stridulous rhonchus.	Pressure of a malignant or aneurismal tumour upon trachea or main bronchus, sometimes produced by laryngeal paralysis.
2. *Moist râles.*	Moist sounds or rattles, produced by the bubbling of air through fluid in the bronchi or lung.	Liquid râles. "Mucous râles" of Laennec.	
Small bubbling râle.	A râle produced by the bubbling of air through fluid in the finer bronchi, and more or less muffled by transmission through spongy lung.	. . . . .	Capillary bronchitis. Pulmonary œdema.
Medium and large bubbling râles.	Similar râles generated in the larger tubes.	Tracheal rattles.	Bronchitis of the larger tubes. Secretion collecting in trachea during last moments of life.

Terms.	Definition.	Synonyms.	Common Significance.
Adventitious sounds (*continued*).			
Small crackling râle.	A fine râle produced in the minute bronchioles of consolidated lung, consisting of numerous small, sharply defined *crackles*, chiefly audible during inspiration, but in less degree with expiration.	Subcrepitant râle.	Thin fluid in minute bronchial tubes, with consolidation of the surrounding lung—*e.g.*, resolving pneumonia.
Medium crackling râles.	Similar to above, but of larger size.	Crepitations. Crepitant râle. Sharp crepitations.	Resolving pneumonia, broncho-pneumonia, or softening tubercle.
Larger crackling râles.	Crackles of larger size, and fewer in number, produced in connection with minute pulmonary cavities.		Softening tubercle.
Gurgling râles.	Larger and more liquid râles, produced in connection with cavities of medium and large size.	Cavernous râles.	Cavity in the lung.
Clicking sounds.	Single sounds, or few in number, mostly limited to inspiration, and of sticky, semi-fluid character.	. . . . .	Commencing softening of tuberculous deposits in the lung.
3. *Fine-hair crepitation.*	A minute dry crackling sound, in which the crackles are infinitely small and even, and occupy chiefly the latter part of inspiration; produced by the separation of sticky alveolar walls which had previously been in contact.	Pneumonic crepitation.	Early stage of pneumonia. The sound is sometimes heard in a certain degree of pulmonary œdema, and during the first two or three deep inspirations over a portion of lung not recently used—*e.g.*, the extreme bases in bed-ridden patients.
Metallic tinkling.	The metallic resonance sometimes imparted to a moist sound by a large pulmonary or other cavity. The sound may be generated in the cavity or resonated from a bronchus in connection with it.	. . . . .	Pneumothorax, or large pulmonary cavity.
Splash.	The succussion of air and fluid in a large cavity, produced by the shock of cough or by shaking a patient, with the ear applied directly to the chest wall.	Hippocratic succussion sound.	A large cavity containing air and fluid. Hydro- or pyopneumothorax.

Terms.	Definition.	Synonyms.	Common Significance.
Adventitious sounds (*continued*).			
Bell sound.	A metallic ring heard on sharp percussion over a pneumothorax, commonly elicited by the use of coins.	*Bruit d'airain.*	Pneumothorax.
Friction.	A rubbing sound produced by the movements of two surfaces of the pleura which are in contact and inflamed.	. . . . .	Pleurisy.
Dry friction.	. . . . .	Leathery or creaking friction. Dry rub.	Heard at commencement and at termination of pleuritic attack.
Moist friction.	A sound often closely imitating moist crepitation, produced by the attrition of surfaces covered by soft, moist lymph.	Spongy friction. Friction crepitus, etc.	Sometimes heard over upper confines of recedent effusion and in other conditions of thick, moist pleuritic exudations.
VOICE-SOUNDS.			
Normal.	The sound of the voice transmitted through the healthy lung.		
Increased or diminished.			
Annulled or absent.	. . . . .	. . . . .	Lung separated from chest wall by fluid or growth.
Bronchophony.	The loud transmission of the laryngeal vibrations, apart from articulation.	. . . . .	Pulmonary consolidation.
Pectoriloquy.	The clear transmission of articulate sounds. Heard best during whispering, when bronchophony (which usually, but not always, accompanies it) is excluded.	. . . . .	Cavity in the lung. A close imitation of the sound—*pectoriloquie aphonique*—may be sometimes heard through a pleuritic effusion (sero-fibrinous) on making the patient whisper roughly.
Ægophony.	A modified bronchophony, having a high-pitched, tremulous character, possibly due to the transmission of the upper tones or harmonics, the fundamental tones being intercepted.	. . . . .	Pleuritic effusion.
Amphoric echo.	An echoing character acquired by the voice sounds when conveyed through a large air-containing cavity.	Metallic echo.	Pneumothorax.

INTERNATIONAL NOMENCLATURE OF PHYSICAL SIGNS.

THE following list of auscultatory sounds was presented by Dr. Austin Flint, Chairman of the Committee appointed at the International Congress in London, in 1881, to report to the Medicine Section of the next Congress on a uniform nomenclature of auscultatory sounds in the diagnosis of diseases of the chest.

The committee nominated were: Dr. Austin Flint, of New York; Professor Ewald, of Berlin; Professor D'Espine, of Geneva; and Drs. Mahomed and Douglas Powell, of London. The subjoined lists were drawn up after much consideration, and after reference to many physicians and teachers of medicine in this and other countries. Although close unanimity existed amongst the members of the committee, the lists were only presented to the Congress in Copenhagen in 1884 as provisional, with the view of eliciting further suggestions and discussion.[4] We may add that an international system of stethoscopic abbreviations was recommended by the Fifth Commission of the International Antituberculosis Association at the Tenth International Conference held in Rome in 1912, and will be found printed in *Tuberculosis*, vol. xi., 1912, p. 488: Berlin, Charlottenburg. It appears to us, however, hardly to fulfil the requirements of British Medicine, and we have not therefore reproduced it.

I. PALPATION.

English and American Lists.	German List.	French List.
1. Vocal fremitus.	Stimmfremitus oder Vocalfremitus.	Vibrations thoraciques.
2. Rhonchal fremitus.	Rhonchofremitus oder Bronchialfremitus.	(Not included.)
3. Friction fremitus.	Pleuritische resp. Pericarditische Reiben.	Frottement.

II. PERCUSSION.

1. Tympanitic resonance.	Tympanitischer Schall.	Son tympanique: (*a*) grave; (*b*) élevé.
2. Amphoric resonance.	Metallklang.	Bruit de pot fêlé.
3. Diminished resonance; dulness.	Dämpfung.	Submatité.
4. Absence of resonance; flatness.	Absolute oder starke dämpfung.	Matité.
5. Increased resonance.*	Hypersonorer Schall.	Son pulmonaire exagéré.
6. Bell sound.	(Not included.)	(Not included.)

III. AUSCULTATION.

A. *First Group.—Varieties of Breath-Sounds.*

1. Exaggerated. Syn.: Puerile; compensatory; supplementary.	Verschärftes Athemgeräusch.	Respiration exagérée.
2. Diminished. Syn.: Feeble; weakened vesicular murmur.	Abgeschwächtes Athemgeräusch.	Respiration diminuée.
3. Suppressed. Syn.: Absence of breath-sound.	Fehlendes Athemgeräusch.	Respiration supprimée.
4. Prolonged expiration: general or local.†	Verlängertes Exspirium.	Expiration prolongée.

5. Interrupted inspiration. Syn.: Jerking, wavy, cog-wheeled.	Saccadirtes Athemgeräusch.	Respiration saccadée.
6. Tubular. Syn.: Bronchial; high-pitched blowing.	Bronchiales Athemgeräusch: (*a*) stark; (*b*) tief.	Souffle bronchique: (*a*) superficiel (*b*) profond; (*c*) doux; (*d*) fort.
7. Vesico-tubular. Syn.: Broncho-vesicular; harsh, coarse, subtubular.‡	Unbestimmtes Athemgeräusch.	Respiration rude broncho-vesiculaire.
8. Amphoric.	Amphorisches Athemgeräusch.	Souffle cavitaire amphorique avec écho musical ou métallique.
9. Cavernous.	(Not included.)	Souffle cavitaire simple ou sans écho musical.
	B. *Second Group.—Adventitious Sounds.*	
1. Rhonchi. Dry musical sounds; (*a*) sonorous; (*b*) sibilant.	Rhonchi. Trockene Rasselgeräusche: (*a*) sonor (schnurrend); (*b*) sibilant (pfeifend).	Rhonchus. Râles secs: (*a*) ronflants; (*b*) sibilants.
2. Stridor.	Stridor. Stenosengeräusch.	Sifflement laryngé. Bruit de cornage; (*a*) trachéal; (*b*) bronchique.
3. Râles. Syn.: Bubbling râles: (*a*) medium; (*b*) large; high or low in pitch.§	Feuchte Rasselgeräusche: (*a*) grossblasige; (*b*) mittelgrossblasige; (*c*) kleinblasige.	Râles humides: (*a*) gros; (*b*) moyens; (*c*) fins; (*a*) avec timbre bronchique; (*b*) sans timbre ou simples.
4. Gurgling.	Höhlenrasseln.	Gargouillement.
5. Clicking.	(Not included.)	Craquements: (*a*) secs; (*b*) humides.
6. Crepitation. Syn.: crepitant râle.	Knisternde Rasselgeräusche; Knisterrasseln.	Râles crépitants.
7. Metallic tinkling.	Metallische Rasselgeräusche; Metallklang.	Tintement métallique.
8. Splash.	Succussionsgeräusch.	Succussion Hippocratique.
9. Friction: (*a*) dry; (*b*) moist.	Reibegeräusch.	Frottement.
	C. *Third Group.—Varieties of Voice-Sounds.*	
1. Increase of vocal resonance.	(Not included.)	Voix exagérée.
2. Diminution or absence of vocal resonance.	Abschwächung und Fehlen der Stimme.	Voix diminuée.
3. Bronchophony.	Bronchophonie.	Bronchophonie.
4. Pectoriloquy.	Pectoriloquie.	Pectoriloquie.
5. Ægophony.	Ægophonie.	Ægophonie.

* American: Increased or vesiculo-tympanitic resonance. † American: Prolonged expiration; high or low in pitch.
‡ American: Broncho-vesicular. Syn.: Vesiculo-tubular.
§ Moist or bubbling râles—(*a*) large, (*b*) medium, (*c*) small; high or low in pitch.

Theory of Percussion.—The exact explanation of the sounds elicited by percussion of the chest in varied conditions is by no means easy, but four factors are concerned in their production. These are—(1) the proportion of air and solid under percussion; (2) the range of vibration of the chest wall; (3) the vibration of the air within the region covered by the chest walls, whether contained in the lung, bronchi, pleura or stomach; (4) the tension of the air within the chest.

The late Dr. C. J. B. Williams and Dr. Bristowe both regarded the percussion note as primarily due to the vibration of the chest wall, and as modified only by the conditions of the underlying cavities or viscera so far as they afford varying degrees of impediment to the chest wall vibration. This view cannot be accepted in its entirety, seeing that percussion of the lung outside the body yields a note closely resembling that of the thoracic sound. It is evident, therefore, that the resonant note over healthy lung must be produced in great part by the vibrations of the air within the lung itself, though these vibrations are doubtless supplemented by those originating in the chest wall. Conversely, in disease, when the lung is rendered solid by inflammation, or compressed by fluid, the vibrations of the chest wall, so far from being reinforced, are damped, and a dull percussion note results.

But although the rhythmic vibrations of the chest are not the only factors in the production of the resonant note of health, as was once held, their importance must not be overlooked, for in disease, and even within the range of health, there are many states in which impairment of resonance is to be accounted for only by a diminished resilience of the chest wall. Thus, many robust people, soldiers especially, have rigid resisting chest walls, and percussion in such persons yields a diminished sound. On the other hand, in children and in delicate persons, with thin elastic ribs, we may obtain an amount of resonance which may even mask a certain degree of underlying disease. Again, in cases of deformity of the chest, such as lateral curvature of the spine, the note over the prominence of the ribs posteriorly is generally, as the late Dr. Hilton Fagge pointed out, much impaired, although the underlying lung be healthy, a fact which can only be explained by the diminished vibrating capacity of the chest wall.

We may now turn to the last factor of which we have spoken, the tension of the air within the chest. This is of less importance than the preceding ones, but, nevertheless, must be taken into consideration. Thus, in the well-formed chest of the young adult, and within the range of normal conditions of quiet breathing, we obtain over the pulmonary regions that degree of resonance and clearness of percussion which is typical of health. At any stage of the inspiratory act, however, the percussion note is somewhat deadened by closure of the glottis and compression of the chest by the expiratory muscles, the tension of the confined air being increased, and its vibratility and that of the chest wall diminished. In pneumothorax, again, with free communication with a bronchus (when, therefore, the air has approximately the normal atmospheric tension), we obtain a deep-toned percussion note, the so-called "tympanitic resonance." If, however, the communication with the lung be only by a valvular opening, admitting the entry, but not the escape, of air, increased tension of the air and of the chest wall results, with corresponding deadening of the note.

Auscultation.

This method of physical examination was not wholly unknown to the ancient Greek physicians, who were in the habit of applying the ear directly to the chest,[5] and had thus discovered the creaking sound of pleural friction, and also the succussion splash. Curiously enough no further progress was made, and with the dark ages the method fell into oblivion, until Laennec, in the early years of the last century, rediscovered it, and worked out its application to disease even in its minutest details. To him, therefore, belongs the honour of being regarded as the true discoverer of auscultation, and the publication of the first edition of his great work, "Traité de l'Auscultation Médiate," in 1819, must ever remain a landmark in the history of medicine.

Auscultation may be practised by using the unaided ear applied directly to the chest, or separated from it only by a towel (*immediate auscultation*), or by employing a stethoscope, either solid or flexible, to convey the sounds from the chest wall to the ear (*mediate auscultation*).

In England the latter method is commonly employed, the binaural flexible stethoscope being now generally used. But it should be remembered that though this instrument possesses great advantages in that it can be used with greater ease and rapidity than the single stethoscope, yet the latter conveys the sounds with greater exactness to the ear, though some of their loudness is lost by their passage through the instrument. In certain circumstances immediate auscultation may be preferred, as when in an advanced case of emphysema the breath-sounds are extremely weak; but the difficulty of localising the exact spot auscultated by the unaided ear militates against the more general adoption of the method, which nevertheless is much practised by certain French physicians of distinction. The student should lose no opportunity of familiarising himself with all three methods of auscultation, and be ready to use each as occasion may require.

Theory of Auscultation.—It is generally agreed that, with the exception of normal vesicular inspiration, all varieties of breath-sound, whether healthy or morbid, are generated in the larynx, and modified in their transmission through the different media which, normally or abnormally, intervene between that organ and the point of observation.

Concerning the vesicular murmur, there has been much diversity of opinion. It has been urged by some that this also is nothing more than laryngeal breathing, altered by its transmission through layers of spongy and badly conducting lung. Others, on the contrary, attribute to the inspiratory portion a double origin, and believe it to be in part a glottic sound, more or less modified by conduction, and in part produced within the lung itself by the formation of a "fluid vein," as the air passes from the terminal bronchus into the expanded infundibula beyond. The latter view seems, on the whole, the more correct, and our knowledge of this subject may be summed up in the following propositions:

1. The vesicular respiratory murmur should be regarded as a sound having a double mechanism, being made up of (*a*) a bruit produced by the passage of air to and fro through the glottis, and reverberated downwards through the bronchial tubes; (*b*) an infinite number of minor bruits similarly produced at the openings of the pulmonary infundibula. The

expiratory portion of the murmur is almost entirely of glottic mechanism.

2. All other breath-sounds are due to the conduction of the glottic bruit (or laryngeal breath-sounds) above mentioned, through media of different kinds, forms and densities; any local currents of air being only so far operative inasmuch as they serve to assist conduction.

Let us now consider the matter in greater detail.

Vesicular Murmur—Normal Breath-Sound.—It was originally thought, as the name, indeed, implies, that the vesicular murmur was produced solely by the entry of air into the air-vesicles of the lung. Very soon, however, this view was opposed, and in 1834 Beau[6] sought to prove that tracheal, vesicular, bronchial and cavernous breath-sounds resulted from the conduction to the surface of the lung of a single sound which was produced in the superior air-passages. The situation at which this sound originates is no doubt the glottis, for here the air passes rapidly backwards and forwards through a small aperture into a larger cavity, giving rise, therefore, to a vibrating column of air, "a fluid vein," and the production of a sound. This laryngeal breath-sound is heard loudly in the trachea, and is traceable down the bronchi as far as they can be followed. It is clear, therefore, that it must enter at least into the formation of the vesicular murmur. And that it does so is proved by the fact that when the larynx is much ulcerated, and the formation of a fluid vein rendered thereby more difficult, or when the organ is entirely eliminated, as by an old tracheotomy and the permanent wearing of a tube, the vesicular murmur over the lung becomes extremely weak. It may be added, also, in support of the part played by the glottis in the origin of the vesicular murmur, that, as shown by Laennec and Beau, in animals with long necks, such as ruminants, the respiratory murmur is much more feeble than in carnivora, though here, possibly, differences in structure and functional activity come into play.

From the above considerations there can be no doubt that the vesicular murmur is to a large extent an altered glottic sound. Is it not, however, in part at least, also pulmonary, produced by the air being thrown into vibration as it passes from the minute terminal bronchi into the larger infundibula of the lung? The chief objection urged against this view

was the belief long entertained that the tidal air did not pass beyond the finest bronchi, and consequently that the currents of air drawn at each inspiration into the infundibula, in obedience to their enlargement with the expansion of the chest, must be so slight and of so low a velocity, as to be incapable of generating a fluid vein and the production of a sound, though the necessary physical conditions are actually present. When we consider, however, the number of infundibula present in the lung (the alveoli have been estimated by the late Professor MacAlister at 355,000,000), it is evident that the infinite repetition of even the very faintest sound must become important, and materially contribute to the respiratory murmur. Further, it was pointed out by the late Dr. Washbourne,[7] in his Croonian Lectures, that it had been demonstrated that the upper air-passages and the ramifications of the bronchial tree possess a capacity of only 140 c.c. and can therefore only accommodate about one-third of the 500 c.c. of air inhaled. The remainder of the tidal air must, therefore, directly enter the infundibula, and, the conditions necessary for the formation of a fluid vein being present, a murmur should arise.

That some portion of the vesicular murmur is actually produced in this way is strongly supported by the observations of Bondet and Chauveau,[8] which have never been contradicted, as well as by the experiments of Dr. J. F. Bullar.[9] The former observers experimented upon a horse suffering from pneumonia, which affected the lower half of the left lung. On auscultating the animal, exaggerated breathing was heard over the right lung and over the upper half of the left, tubular breath-sound being distinct over the lower or affected half of the left lung. These preliminary observations made, the trachea was opened by an incision twenty centimetres in length, and the following phenomena were noted:

1. On auscultation over the trachea below the incision, the wound being held widely open, the inspiratory bruit was almost completely lost, and the expiratory sound but faintly audible.

2. On auscultation over the consolidated lung, whilst the wound in the trachea was held widely open, *no tubular sound could be heard during inspiration, and only a faint, brief, and*

abortive sound during expiration. Over the rest of the lung and over the opposite (sound) lung, however, the *normal respiratory sounds were clearly heard.*

It should be added that a musical reed was introduced into the trachea, and that the artificial voice-sound so produced, though inaudible over the healthy lung, was heard distinctly over the consolidated area, thus proving that a possible source of fallacy—viz., the blocking of the left lower bronchus by blood-clot—did not exist.

It would seem, therefore, from these considerations, that the pulmonary element does take an important share in the production of the normal respiratory sound, and is, indeed, the cause of its characteristic quality.

Abnormal Breath-Sounds.—*Weakness of breath-sound* may simply arise from feebleness of the muscular chest movements. Often, however, it is the result of disease, and is perhaps best observed when a small pleural effusion partially compresses the lung. In emphysema, also, when the expansion of the lung is impaired owing to the over-distension and atrophy of its tissue, which characterises the disease, the respiratory sounds are weakened.

Suppression of Breath-Sounds.—When a lung is separated from the surface, and compressed by a sufficient layer of fluid, or has its bronchus occluded from any cause, suppression of the breath-sounds follows. Even in cases of considerable effusion, however, a certain amount of breath-sound is heard over the upper part of the chest, the fluid naturally gravitating to the lower portion of the thorax, so that the upper area of the lung escapes compression. When the lung is separated from the thoracic wall by thickened adhesions, and, as is usually the case, is indurated and collapsed, the breath-sound is partially suppressed, the glottic bruits being only very imperfectly conducted.

Exaggerated Breathing.—This is heard in the healthy child. More rapid breathing, more rapid passage of air through the glottis and into the lungs, and greater thinness of the chest walls, are the conditions upon which this enhanced breath-sound depends. The expiration is generally rather prolonged, and both sounds are coarser than ordinary. Exaggerated breath-sound is usually, in health, limited to children of tender years, and hence its synonym "puerile breathing," but some

persons preserve this quality through adult life. This variety of breathing is heard over the sound side in cases in which the respiratory function of one lung is in abeyance from any cause. It may be similarly heard over one portion of a lung, the remainder of which is diseased. Hence another synonym, "compensatory breathing." It indicates respiratory vigour, and is so far of good augury, inasmuch as it shows that an enlarged lung, or portion of a lung, is not merely dilated, but also functionally more active—a most important fact to ascertain in cases of one-sided chest disease.

Jerking, Wavy and *Cogged Breathing* ("cog-wheeled respiration") are varieties to which different authors have attached very different importance. In jerking or wavy breathing the inspiration, instead of being a continuous sound, is two or three times interrupted. The expiration is rarely affected in this way, and may be normal, or simply harsh and somewhat prolonged. One so frequently meets with jerking respiration in nervous people that, when unaccompanied by other morbid sounds, but little importance should be attached to it. The following are some of the commoner abnormal conditions under which it may occur: (1) It may be due to irregular action of the respiratory muscles under conditions of pain, as in pleurodynia, pleurisy, and myalgia. (2) It may be the result of hysteria and other nerve disorders. (3) It may be produced (mostly on the left side) by cardiac pulsation, its rhythm being then synchronous with that of the heart. (4) In certain cases it may be accounted for by textural diseases of the lung itself, or by pleural adhesions, which lead to a want of uniformity in pulmonary elasticity and expansile power. For this reason it may sometimes be met with in phthisis at quite an early stage of the disease. A remarkable instance of wavy cavernous breathing was observed by one of us in a case in which the entering bronchus to a large cavity was partially occluded by a small pulmonary aneurism projecting into its lumen close to the cavity, and thus alternately stopping and permitting the cavernous sound. The death of the patient from sudden hæmoptysis, a few hours later, verified and explained the observation.[10]

Prolonged Expiration.—Prolongation of expiration is perhaps the most important, as it certainly is the earliest of the signs of commencing consolidation of the lung. It marks

the transition between vesicular and bronchial breath-sound, and it indicates that over a certain extent of lung the factors, which normally lead to the production of the vesicular sound, are partially, at least, in abeyance, tending therefore to the predominance of the unaltered glottic sounds, with their distinctive characters and proportion. Thus, with prolongation of the expiration are associated other characteristics of the glottic element. For example, division between inspiration and expiration (*divided respiration*) may be often noticed; and as the expiration becomes more prolonged, equalling, or even slightly exceeding, the inspiration, the vesicular quality of the breath-sounds gradually disappears, the breathing acquiring at first a harsh, and later a true bronchial character. Indeed, all stages between simple prolongation of the expiration and true bronchial breathing, in which the glottic sounds are conducted in all their intensity, may be observed. To certain of these intermediate stages the terms "harsh," "vesiculo-bronchial," "transitional," or "indeterminate," have been applied.

Bronchial Respiration.—This variety of respiratory murmur is heard normally at the lower part of the trachea, and in less degree in the first intercostal space for a short distance on either side of the sternum. In many subjects, especially, as Laennec pointed out, those who are very thin, it may be heard over the interscapular regions, from the last cervical vertebra to the level of the third or fourth dorsal, owing to the proximity of the trachea and large bronchi in this region. As a morbid sign it is heard in its most characteristic form over a lung consolidated by acute pneumonia. Bronchial breath-sound possesses a peculiar penetrating and blowing character, and, as Skoda pointed out, can best be imitated by placing the tongue in the position to form the consonant "ch" (hard χ), and drawing the breath to and fro. It differs, also, from vesicular breathing in that expiration is often as loud and long as inspiration, and that the two stages of respiration are separated from each other by a perceptible pause. It varies in intensity according to the degree of consolidation and the permeability of the bronchial channels.

Laennec was of opinion that the sounds originated in the affected lung, but it is now admitted that they are in reality generated in the larynx, and thence conducted down the air-

passages into the chest, and so to the observer's ear. The following are the reasons for this belief:

1. The breath-sounds heard over the larynx, and produced there, possess all the characteristics of bronchial breathing, and differ from the latter chiefly in their greater intensity.

2. In pneumonia, in which this form of breathing is especially heard, the lung is impacted by a coagulated substance which fills it, and maintains it in the state of permanent inspiration, a condition which forbids the entry of air. The formation, therefore, of a fluid vein and the production of a sound within the affected lung is, under these circumstances, impossible.

3. The experiment of Bondet and Chauveau upon the hepatised lung of the horse, to which we have already referred, points strongly in the same direction. These observers, on holding widely open the wound in the trachea, caused the respiratory bruit heard over the hepatised lung to disappear, whilst over the sound lung the respiratory murmur was scarcely at all modified.

It would appear, then, that the two conditions for the production of bronchial breath-sound are consolidation of lung and patency of bronchi. Any obstruction of the main bronchus will obscure the sound, and, we may add, separation of the consolidated lung from the parietes, whether by fluid or thickened pleura, will render it less distinct. In some rare cases of pneumonia the breathing is suppressed owing to the exudation overflowing from the alveoli into the small bronchi, and thus occluding them. Bronchial breath-sound may be produced by other than diseases of the lung. A mediastinal tumour, whether aneurismal or cancerous, situated between the thoracic wall and a bronchus, will yield the sound, the vibrations within the tube being conveyed through the substance of the tumour almost unaltered to the chest wall.

By many writers the term *tubular* is used as synonymous with bronchial, tubular and bronchial breathing being thus interchangeable names. Some, however, restrict the term "tubular" to that variety of bronchial breathing which is high-pitched and whiffing in character, such as is often heard over a pulmonary cavity.

Laryngeal and *Tracheal Breathing*, heard on auscultation over the larynx and trachea respectively, possess in a marked

degree, as Laennec pointed out, the characteristics of bronchial breathing, and differ only in the greater loudness and intensity of the sounds.

Cavernous Breath-Sound.—This consists of a hollow inspiratory and expiratory sound, which has been compared to the sound produced by blowing into the cavity formed by the two hands. The expiration must especially be attended to in listening for this variety of breath-sound; it is wavering, hollow, and prolonged. We agree with the late Dr. Austin Flint in attaching importance to the fact that the expiration is of lower pitch than the inspiration, as serving to distinguish cavernous respiration from bronchial, in which the inspiratory and expiratory are of similar pitch. Cavernous breathing is heard over pulmonary cavities, which are of a certain size (larger than a filbert), which are in part at least empty, and which communicate freely with one or more bronchi.

As to the mechanism of this sound, it cannot be denied that it may be imitated by breathing into the hollow of the two hands, a fact which suggests that within the chest it may be produced by the air entering and leaving a large cavity. But it may be stated at once that in general this is not the case. Cavernous breathing is nothing more than bronchial breathing modified by the resonating properties of a large cavity; but though the vibrations which produce it must pass from the bronchus to the cavity, to be there modified and acquire that reverberating, "cavernous" quality which distinguishes this form of breathing, it is not necessary for the air itself to enter, nor, indeed, would this be always possible, owing to the unyielding character of the cavity walls. That the sound is not dependent upon such entry is further supported by the fact that the cavernous quality is heard very loudly during expiration, whereas the expulsion of air from a cavity could not give rise to the formation of a "fluid vein" and the production of a sound. In short, cavernous breathing is produced by the conduction of the laryngeal vibrations into a cavity, and their consequent modification, the entry of air into the cavity not being an essential factor in the process.

Amphoric Breathing.—This is really a variety of cavernous breathing, the cavity being large, superficial and thin-walled. It may be imitated by breathing into a large jar or amphora,

whence the derivation of the name. In cases of pneumothorax in which the opening is free, this form of breath-sound is heard to perfection, but, as with cavernous breathing, penetration of the air into the cavity is not essential (Gee).[2b]

Adventitious Sounds.—With regard to the various adventitious sounds heard within the chest, we have based our classification upon that originally suggested by Laënnec. The term "râle" is thus retained as a general name to include the numerous added sounds which in disease are produced within the air-passages and lungs by the act of respiration.

Of râles there are three chief varieties:

1. The dry râle, with its subdivisions, rhonchus and sibilus.
2. The moist or liquid râle—"mucous râle" of Laennec—with its subdivisions, bubbling, crackling, gurgling and cavernous râle.
3. The fine-hair crepitation.

In listening for these adventitious sounds, our attention will be attracted mainly to the inspiratory portion of the respiratory act, during which they are as a rule best heard; whilst modified breath-sounds are most characteristically heard during expiration.

Concerning the mode of formation of these sounds, some are simple, and require but little explanatory comment. *Rhonchi* and *sibili* are but musical notes of higher or lower pitch, depending upon vibrations of air set up in tubes partially obstructed by local thickenings of their lining membranes, by mucous collections or pellets adhering to their sides so as partially to obstruct them, or, finally, by spasmodic narrowing of the tubes themselves. It will be observed that all these causes of sibilant and sonorous râles within the chest are of a temporary or transient nature, and accordingly these sounds are very inconstant, appearing and disappearing, or shifting from one spot to another, even whilst the patient is under examination. Thus, a vigorous cough, clearing the tubes of mucous collection, will often for a time remove a sonorous rhonchus, while the sibilant sounds in asthma, or bronchitis with spasm, will be partially or wholly removed by the inhalation of chloroform or ether. It must further be noted that in all cases the true respiratory murmur is obscured and enfeebled by the presence of these rhonchi, to return on their removal; and it is a matter of great impor-

tance in practical auscultation to observe whether the respiratory murmur is altogether thus obscured, or whether the air penetrates more or less through the râles into the vesicular tissue.

Stridor is a variety of rhonchus in which the vibrations are very coarse, and the sound produced rough and low-pitched. It is best heard during inspiration. It may be occasioned by some paralytic affection of the glottis, which prevents the cords from being drawn apart during inspiration, and allows them partially to flap together, and thus to vibrate. It is in other cases produced by the pressure of a tumour directly upon the trachea or a main bronchus. The vibrations which produce stridor, and those of the stronger forms of rhonchus, are so coarse as to be perceptible to the hand applied to the surface of the chest, thus producing *rhonchal fremitus.*

Liquid or Moist Râle.—This variety of râle, described by Laennec under the heading "Mucous Râle," is produced, as he pointed out, by the bubbling of air through fluid, of whatever nature, contained either in the bronchi or in cavities in the lung. The sound may be exactly imitated by blowing into soapy water with a pipe or straw. These râles differ in character according to the bubbles which produce them, and were accordingly classified by their discoverer into "large," "medium," and "small." They are much modified also by the condition of the surrounding lung. Thus, if the air-vesicles around are healthy, as in bronchitis, they have a "bubbling" quality. If, on the other hand, there is more or less consolidation, they acquire a more sharply defined character, and are spoken of as "crackling" râles. If, again, they are formed in connection with a cavity, they become echoing and reverberating, and are termed "gurgling" or "cavernous."

The *Crackling Râle*, "humid crackling," "humid clicking," or "sharp crepitation," is significant of fluid in the tubes, together with the presence of more or less consolidation in the surrounding lung. This râle is heard in broncho-pneumonia, and very frequently in the softening stages of phthisis, in which the bronchial tubes are filled with liquid débris from the softening foci, and are themselves surrounded by patches of solid lung. In character it is a sharply-defined, moist

sound, consisting of two or three inspiratory and one or two expiratory elements, the respiratory murmur being itself often masked or replaced by the crackles. The sound varies much in sharpness of definition, according to the condition of the pulmonary tissue. If this be in a state of massive tuberculous consolidation, as in the most acute form of phthisis, the clicks are strongly conducted, and metallic in character. In other cases, when the softening nodules are more widely separated by spongy and tolerably healthy lung tissue, the crackles are muffled and more or less obscured. Over portions of lung which are functionally inactive under ordinary circumstances of respiration, owing to fibroid change or pleural thickening, no moist sounds may be heard when softening takes place until a sharp cough has forced air into the tissues and produced them. This is a most important point to remember in auscultation.

Cavernous Râle—Gurgling Râle.—This is nothing but a crackling râle of large size, and of a loud, reverberating quality. Bubbles bursting in a cavity will no doubt account for the formation of such sounds, as we may perceive on listening over a secreting cavity whilst the patient coughs, when we hear the splashing and gurgling sounds resulting from the forcible commingling of air and fluid. But in large and comparatively dry cavities we can hardly imagine any such commingling to take place during ordinary or deep breathing, even supposing that air passes backwards and forwards into the cavity with any appreciable current, which is not necessarily the case. In truth entry of air into the cavity is not essential to produce the sound, since any moist râles in the associated bronchi will be heard with exaggerated intensity over the cavity, and in this way no doubt many gurgling or cavernous râles are produced.

Should the cavity be of sufficient dimensions, and the râle very fine, it may then take on special characteristics, and acquire the qualities of *metallic tinkling*, a sound aptly compared by Laennec to that produced by striking a metal vessel with a pin. It is entirely erroneous to suppose—as may be demonstrated frequently on post-mortem inspections—that this variety of râle is significant of pneumothorax only; it may be well heard over a large cavity, and its mechanism is probably the same in both instances—viz., the reverberation

through a large, thin-walled, and empty cavity of a very fine moist râle produced in the bronchus or fistulous channel leading to the cavity.

Fine-Hair Crepitation.—Great difference of opinion has prevailed respecting the nature and mechanism of this variety of râle. All observers agree that it is best heard in the first stage of pneumonia, and some regard it as the pathognomonic sign of that disease. This sound may be defined as composed of a variable, sometimes immense, number of sharp crackling sounds of minute size, all perfectly similar to each other, and rapidly evolved in puffs more or less prolonged. The sounds are dry in quality, and coexist exclusively, except in rare cases, with inspiration, and, once established, remain persistent until superseded by other phenomena. The late Dr. C. J. B. Williams's description of the sound, as resembling that produced by slowly and firmly rubbing a lock of hair between the finger and thumb close to the ear, has been generally accepted.

The fine-hair crepitation may, however, occur in conditions other than pneumonia. Thus it may be heard in healthy chests, when we auscultate the bases of the lungs of those who have been confined to bed, and instruct them to take a deep breath, thereby opening up alveoli which have been for a time disused. In disease it may occur in cases of pulmonary collapse, in certain degrees of pulmonary œdema and in the first stage of pulmonary apoplexy; although it must be confessed that in these conditions the sound does not possess that sharpness which characterises it in pneumonia, and which is attributable to the increasing density of the surrounding lung. Whereas in pneumonia the sound is accompanied by bronchial breathing, in other conditions it is as a rule unattended by any breath-sound. The sound is apparently produced by the separation of sticky aveolar walls which have previously been in contact, and in pneumonia the actual state of the lung which gives rise to it may be assumed to be one of œdematous exudation from inflammatory congestion. When the exudation into the air cells is complete and consolidated, the crepitation ceases.

A reference may be made to one other variety of râle described by Laennec, the *Dry Crackle*, "craquement ou râle crepitant sec à grosses bulles." This was believed by

him to have a special character, and to be pathognomonic of emphysema. In this view he has not been confirmed by later observers. The term has long been in use at the Brompton Hospital as descriptive of the few inspiratory crackles regarded as significant of commencing tuberculous softening.

Pleural Friction.—This sound has been known from the earliest times, having been described by the Hippocratic writers, who compared it to "the grating or creaking of a leather thong."* Curiously enough its significance was misunderstood by Laennec, who believed the "bruit de frottement ascendant et descendant," as he termed it, to be the result of intralobular emphysema.[11] Its true relation to pleurisy was demonstrated a few years later by Reynaud.[12]

Friction is produced by roughening and inequalities of the pleural surfaces, so that the two layers no longer glide smoothly upon each other. It is well heard, therefore, in the early stages of pleurisy, when the pleuræ are roughened, but still in contact. The sound may occur over any area of the chest, but is best heard over the lower portions, since here the movements of the pleural surfaces are most extensive, owing to their proximity to the diaphragm. In character the sound may resemble the creaking of a piece of leather, *dry friction*, or it may be softer in character and more like a moist râle, the term *moist friction* being then applied to it. Friction may accompany both inspiration and expiration, and it is usually best heard at the end of inspiration and the commencement of expiration. If the pleura in contact with the heart be inflamed, the sound may be synchronous with the beat of the heart, and suggest pericarditis. *Pleuro-pericardial friction*, however, as the sound is then termed, may be distinguished from true pericardial friction by the influence upon it of the respiratory movements, a deep inspiration or a deep expiration materially modifying the sound.

Voice-Sounds.—Enough has been said in the earlier part of this chapter respecting the mechanism of breath-sounds to make it clear that there can be no good reason for any mystery as to the way in which voice-sounds are produced,

* "If the lung falls against the side, the patient coughs . . . a heavy weight seems to exist in the chest, and sharp pains sting him; and it grates like a leather thong, and stops the breath" (Hippocrates, "De Morbis," ii., § 59).

or in which they are intensified or annulled under different morbid conditions of the lungs and pleuræ. There can be no doubt that they are produced in the larynx, and conducted thence downwards through the bronchial tubes. These channels in the normal state are held patent by the elastic traction upon them of the surrounding lung, and they serve, therefore, as so many speaking-tubes, although the direction of the current of air during articulation is not favourable to the best possible conduction. The rapid subdividing of the tube and the embedding of the branches in an ill-conducting material is, moreover, highly unfavourable to conduction of sound, and were it not for the shortness of distance, the voice-sounds would not be audible at all. As it is they are conducted to the ear in a muffled manner, and to the buzzing sound so produced the term *normal vocal resonance* is applied.

If the glottis be destroyed by tuberculous or syphilitic disease, the voice-sound is, of course, annulled, and even the whisper sound is impaired, showing that the glottis is also concerned in its production. If the main bronchus on one side be compressed or occluded, the voice-sounds are annulled on that side, and vocal vibrations, which are merely palpable sounds, are similarly obscured. A like result also follows if the pleura be filled with fluid, or the chest walls be very fat, both conditions acting as a barrier to the transmission of the vibrations. If, on the other hand, the termination of the tubes be surrounded by a better conducting material, as in pneumonia, the sounds will be conducted to the surface of the chest with their laryngeal qualities but little altered, and they will be heard loudly and clearly as though one were listening directly over a bronchus. *Bronchophony*, in short, will be produced. If, again, a large bronchus, instead of being subdivided into innumerable smaller and diminishing branches, abruptly terminates in an empty cavity occupying the area of its former distribution, then the voice and whisper sounds transmitted downwards will be no longer muffled by the normal lung tissue, but will be loudly heard over the region of the cavity, giving rise to bronchophony and *whispering pectoriloquy*.

In certain cases of pleural effusion, and sometimes in pneumonia, the bronchophony has a high-pitched, nasal and tremulous character, and is then spoken of as *ægophony*. The

late Dr. Stone advanced the view, which has since been generally accepted, that the peculiar character of this voice-sound is to be ascribed to the suppression or partial suppression of the fundamental tones, and the accentuation of the higher overtones or harmonics. He further believed that for this change to take effect the presence of fluid is essential, the slower vibrations being unable to penetrate the liquid, while the more rapid vibrations of the higher notes are transmitted.

This view, in so far as it presupposes the presence of fluid, has been disputed on experimental grounds by Sir Frederick Taylor,[13] who suggests that in all cases the change is produced *by the compression of the bronchial tubes*, whether by fluid or by some exudation into the alveoli, so that their shape becomes altered, and whereas normally they resonate the lower harmonics, now they can resonate only the higher ones. The tremulous, bleating character of the sound he would regard as simply the result of discord produced by the presence of "beats" occurring between the higher harmonics thus reinforced. The hypothesis is an interesting one, especially as it attempts to explain what is undoubtedly a fact—namely, the not uncommon occurrence of ægophony in cases of pneumonia, quite apart from any effusion into the pleural cavity. It is to be remarked, however, that in cases of moderate effusion, in which the ægophony is heard to perfection, the bronchial tubes are not compressed, as we have elsewhere pointed out.

Amphoric or *metallic echo* is a voice-sound which is often heard in cases of pneumothorax. It is probably to be regarded as a reflected sound, due to the impingement of vibrations against the walls of a large echoing cavity. It is best heard in cases in which the communication with the pleura is very small.

Auscultatory Percussion.—*The Bell Sound*, or *bruit d'airain*, is a physical sign which we really owe to Trousseau,[14] though it is probable that Laennec was to some extent cognisant of it. It is elicited by auscultation over a large cavity containing air, while an assistant percusses with two coins over the same region. The ringing sound thus produced differs markedly from the dull thud which alone is heard over the adjacent lung. In cases of pneumothorax the bell sound is often heard to perfection; but it may also be heard over large,

smooth-walled, pulmonary cavities, or a distended stomach. Subphrenic abscesses or hydatids with decomposing and gaseous contents may also give rise to it.

The peculiarly ringing and musical quality of the sound arises from the air in the cavity responding to, and vibrating in unison with, one of the tones comprised in the sound produced by striking the coins. The note being thus "resonated" or reinforced, and the vibrations following each other at regular intervals, a musical character results. The fact that the bell sound is not always heard over a pneumothorax, or, having once been heard, may disappear, is probably to be explained by alterations in the size of the cavity (fluid tending to replace the air), or by variations in the tension of the contained air. In this way the fundamental tone of the cavity becomes altered, and reinforcement of the coin sound is rendered difficult.

The combination of auscultation and percussion just described has been employed for very many years, but more recently a modification of the method has been introduced with the hope of ascertaining more exactly the limits of the organs within the chest, and especially of the solid organs, where these are in contact with other substances which also yield a dull note to percussion. The method is based upon the belief that the vibrations of the struck coins, or of a tuning-fork placed upon the chest wall, are conducted over the chest partly through the parietes and partly through the underlying viscera, and it is asserted that, so long as the stethoscope is confined to the organ over which the pleximeter coin or tuning-fork is placed, the sound is heard with comparatively little diminution in loudness; but so soon as the limit of the viscus is overstepped, or we pass from one lobe of the lung to another, a corresponding hindrance to the passage of vibrations occurs, and the sound at once loses much of its intensity. We have tested this method, but have not been able to satisfy ourselves of its accuracy.

Roentgen Rays.

After examining the chest in the manner which we have described, it is sometimes advisable to supplement our methods by the employment of the X-rays. For the application of Professor Roentgen's discovery to the diagnosis of

diseases of the chest we are much indebted to the pioneer work of Dr. Hugh Walsham. The patient may be examined by means of the screen (radioscopy), or a photograph may be taken (radiography). With the former, in addition to the shadows which alone are represented in a photograph, a lightening and deepening of the shadows during inspiration and expiration respectively can sometimes be observed; the movement of the heart and diaphragm can be also studied, as well as the pulsation of any abnormal tumour which may happen to be present. But in a doubtful and difficult case a photograph (radiogram) should not be neglected, since shadows are often thus demonstrated with greater distinctness.

In certain chest diseases the rays prove of great assistance. Thus, in a case in which the diagnosis lies between aneurism and new growth, an examination with the screen may reveal the sharply-defined pulsating shadow so suggestive of the former. Similarly, the presence of a foreign body in the chest may be shown, and its situation accurately localised by the stereoscopic method invented by the late Sir Mackenzie Davidson. Again in hydatid disease of the lung the X-ray appearances are often characteristic.

In pneumothorax the appearances seen are instructive and interesting, but, as ordinary clinical methods are generally sufficient for the diagnosis, the rays are here of less assistance. In regard to the early diagnosis of phthisis, symptoms and physical signs have in our experience manifested themselves as a rule before any characteristic X-ray appearances.

Whilst thus fully admitting the value of X-ray examination as an additional aid in the diagnosis of certain affections of the chest, we must add that we have found its results as liable to fallacy as other methods of physical examination, and it is only to be regarded as one amongst other modes of diagnosis, including the history, symptoms, and auscultatory and percussion signs, in the complete investigation of any difficult case. To the appearances seen in the various forms of chest disease we shall refer in greater detail in the succeeding chapters.

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CHAPTER IV

EXAMINATION OF THE SPUTUM

An examination of the contents of the spittoon should never be omitted in chest illness, since it may give valuable aid in diagnosis.

The sputum is derived chiefly from the following sources:

1. The buccal cavity, yielding saliva, débris of food and occasionally blood.

2. The naso-pharynx—in conditions of catarrh.

3. The trachea and bronchial tract—in tracheitis, bronchitis, bronchiectasis and asthma.

4. The lung proper, under the following conditions:—

> (*a*) inflammation or œdema of the infundibular and alveolar spaces, as in pneumonia and œdema of the lung; (*b*) suppurative or other destructive changes in the lung—as in abscess, phthisis, hydatid, or growths; (*c*) the rupture of vessels or of an abscess or other collection into or through the lung, as in aneurism, empyema or hepatic abscess.

Under each of these conditions the expectoration is to some extent characteristic, and it is therefore obvious how varied must be the appearances of the sputum, and how important the information to be gained by its careful and minute examination—similar, indeed, to that obtained from the completely analogous sedimentary deposits from the urine in kidney and bladder diseases.

It is enough here to have alluded to the main sources whence varied forms of sputum are derived, leaving more detailed descriptions to be given in the chapters dealing with the diseases referred to. We propose, however, briefly to describe the most appropriate methods of examining sputum, and more especially to speak of its microscopical investigation.

The quantity, reaction, consistence, colour and transparency of the sputum should be carefully noted.

Quantity.—This varies considerably, in some cases being scanty, in others very large.

The largest amount is brought up in hæmoptysis, acute pulmonary œdema, or when an empyema bursts through the lungs. In bronchiectasis also the expectoration is often very copious, at times seeming almost to pour out of the mouth of the patient, and in certain forms of bronchitis (bronchoblenorrhœa) the quantity is large. In phthisis, too, when cavities exist, a great amount of muco-purulent material is expectorated.

Reaction.—The reaction of sputum is always alkaline. When an acid reaction is obtained, contamination with vomited matters should be suspected.

Consistence.—According to the varying proportions of mucus and pus present, the sputum assumes a mucoid, muco-purulent or purulent form. Each variety may, however, from time to time occur in the course of any one disease.

Mucoid expectoration is glassy and transparent, and may be quite watery, owing to a large amount of saliva being mixed with it. A typical example of mucoid sputum occurs in the early stage of bronchitis. Muco-purulent expectoration may be of any consistency; there may be only a few white flakes floating in it, or it may consist mostly of pus.

Stratification is not infrequently noticed, this being best observed when the contents of the spittoon are emptied into a conical glass. After some hours three layers will have formed, the uppermost of which is semi-opaque, greenish-yellow and frothy, containing small masses and balls, hanging down into the second layer, which is more transparent and watery. The undermost layer, composed of pus cells and decomposing débris, is opaque and of a dirty yellow colour. Stratification is best seen in cases of bronchiectasis.

In phthisis, especially where there are moderate-sized cavities, the sputum commonly forms flattened lumps of rounded smooth outline floating on the fluid in the spittoon. Each of these *nummulated* masses consists of a collection of pus which has been expelled from a cavity, and has become moulded and clothed with mucus as it passes through the bronchial tract, thus forming a separate mass, which assumes a flattened shape

when floating on water. This variety is sometimes also met with in bronchiectasis.

Purulent expectoration, consisting of almost pure pus, occurs only when an abscess in the lung has broken through into a bronchus, or an empyema has burst into the lung.

Serous sputum is met with in connection with acute œdema of the lungs. It separates into an upper frothy and a lower fluid layer, which partially or completely coagulates on heating.

Colour.—When the sputum consists only of mucus and a few cells, it is almost clear. If pus is present, it becomes white and more opaque; and if the quantity of pus be large, a greenish tinge is often noticed.

The chief differences in colour are caused by the addition of blood or blood-colouring matter. The subject of hæmoptysis will be more fully considered in a later chapter; the more remote changes which blood sometimes undergoes need only be mentioned here.

In pneumonia the sputum has generally a rusty tinge, due chiefly to dissolved hæmoglobin, as red corpuscles are present in too few numbers to produce the colour. In rare cases, quite apart from any jaundice, the pneumonic sputum may acquire a grass-green colour. This generally results, as Von Jaksch[1] has shown, from the conversion of the hæmoglobin or hæmatin into bilirubin, and the oxidation of the latter into biliverdin. Occasionally grass-green sputum has been observed in other diseases, such as asthma, and may then be traced, as pointed out by Rosenbach,[2] to certain pigment-forming micro-organisms of no pathological importance. Towards the end of the pneumonic attack the rusty colour gives place to a saffron or citron-yellow tinge, the same colour having been observed also in rare cases of pulmonary tuberculosis. Occasionally, in cases of pneumonia which run an unfavourable course, a frothy, watery, dark red fluid, known as *prune-juice* expectoration, is coughed up. This is sometimes also observed in sarcoma or cancer of the lung.

In cases of tropical abscess of the liver discharging through the lung, the expectoration assumes a very characteristic appearance, and has been likened to anchovy sauce. The colouring matter is derived in this case from the presence of red corpuscles, crystals of hæmatoidin, and disintegrated liver

cells, which give the reddish-brown, brick-dust colour. The amœba of dysentery is nearly always present.

Occasionally in gunshot wounds of the chest involving the lung and liver, bright yellow bile may be met with in the sputum.[3]

In certain manufacturing districts iron-dust may tinge the expectoration an ochre yellow or red colour, while among coalminers it may be almost black from the inhalation of particles of carbon. From a similar cause the sputum of those who dwell in large towns is always more or less darkened.

Bronchial Casts.—Casts of the bronchial tubes, composed in most cases of fibrin, with more or less numerous cellular elements embedded, are sometimes coughed up. These are occasionally expectorated after an attack of hæmoptysis, and are then found to consist of fibrin enclosing in its meshes numerous red blood-corpuscles. Their red colour will distinguish these casts from other varieties. Usually such casts are small in size, branching in character and measure perhaps a couple of inches in length. Occasionally, however, they are very much larger, as in the beautiful example, apparently of this nature, figured in the "Observationes Medicae" of Nicholas Tulp.[4] We have never seen a cast of such unusual size expectorated during life, although we have removed after death following hæmoptysis blood-casts from the air-passage of which Fig. 10 illustrates an unusually complete example.

Casts consisting of complete mouldings of the trachea and large bronchi are still sometimes seen in cases of diphtheria, and were of common occurrence before the introduction of antitoxin. More often they are smaller and have their origin in the second and third divisions. In pneumonia, casts of the finest bronchi are not infrequently seen, if search be made for them, but they always remain small in size, since the inflammatory process does not spread beyond the finer tubes. It is, however, in that rare disease "plastic" or "membranous bronchitis," which we shall consider in detail in a later chapter, that the most perfect bronchial casts are to be observed.

Microscopic Examination.

In order to make a satisfactory examination of the sputum with the microscope, the selection of the sample is important, all food materials being so far as possible excluded. For this

purpose the morning expectoration, collected before any food has been taken, is most suitable for examination.

The sputum should be poured on to a plate, the black vulcanite dishes used by photographers being well adapted for the purpose. Small particles should be picked out from the general mass by means of forceps and scissors, placed on a glass slide, and gently flattened out with a thin cover-glass.

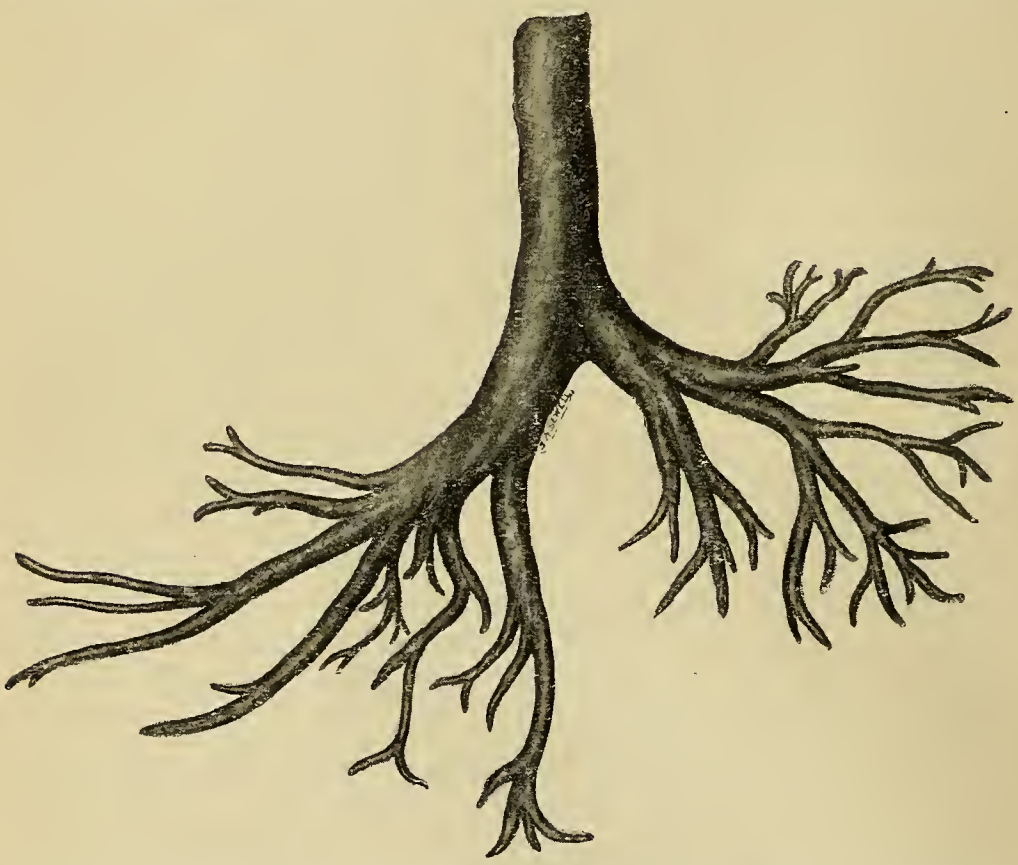


FIG. 10.—SHOWING A LARGE BLOOD-CAST FOUND AFTER DEATH IN THE AIR-PASSAGES OF A PATIENT WHO HAD DIED FROM HÆMOPTYSIS (REDUCED TO $\frac{2}{3}$ NATURAL SIZE).

(From the Brompton Hospital Museum.)

The most opaque portions should generally be selected, in which may be observed:—

Red Blood Cells (Fig. 11, *g*), the individual corpuscles of which usually preserve their colour and discoidal form, though in pneumonia they are often seen as pale colourless rings. If the blood has been retained for some time in the lung, the corpuscles may disappear, and be represented by crystals or irregular particles of hæmatoidin (Fig 11, *e*).

Leucocytes and Pus Cells (*Fig.* 11, *f*, *f'*).—These are present to some extent in all sputa, and if the expectoration be very

purulent it may be almost composed of them. The variety chiefly found is the common pus cell or polymorphonuclear leucocyte. The cells frequently enclose granules of fat and particles of pigment; on addition of acetic acid the tripartite nucleus becomes well defined. In many cases of asthma, and in some of bronchitis, considerable numbers of coarsely granular eosinophile cells may be observed.

Epithelium.—The epithelial cells found in sputum are of three kinds, squamous, ciliated or columnar, and alveolar.

1. *Squamous Cells* (Fig. 11, *h*) are derived from the mouth, pharynx, and upper part of the larynx. They are always

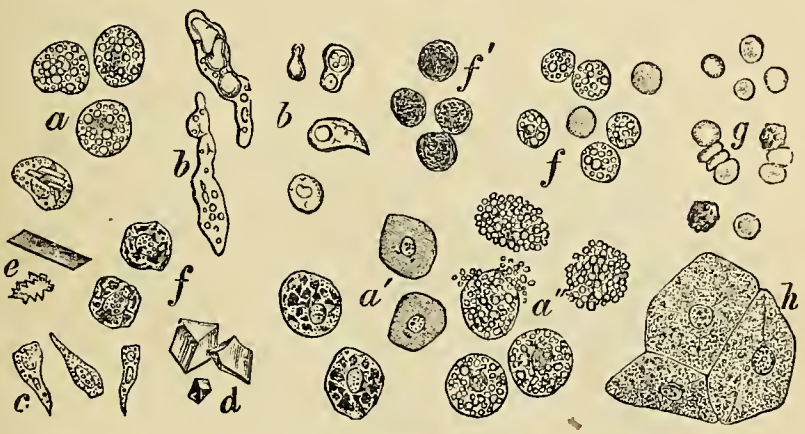


FIG. 11.—SHOWING EPITHELIUM, LEUCOCYTES, AND CRYSTALS FOUND IN SPUTUM (AFTER VON JAKSCH).

a, *a'*, *a''*, alveolar epithelium; *b*, myelin forms; *c*, ciliated epithelium; *d*, crystals of calcium carbonate; *e*, hæmatoidin crystals and masses; *f*, *f'*, white blood-corpuscles; *g*, red blood-corpuscles; *h*, squamous epithelium. (Eyepiece III., Objective 8a, Reichert.)

present, and have no pathological value. When the sample is very liquid, clear, and contains only these cells and no other form of epithelium, it may be concluded that the specimen consists of saliva only.

2. *Ciliated or Columnar Cells* (Fig. 11, *c*) are derived from the greater part of the respiratory passages, including the posterior nares. When seen in large numbers, they indicate a catarrh of the passages. Cilia are only found attached to them when the inflammatory process is very acute.

3. *Alveolar Cells* (Fig. 11, *a*, *a'*, *a''*) from the air-vesicles of the lung are but rarely seen in a perfect condition. They are elliptical in shape, each containing one nucleus, which usually requires acetic acid to render it visible. The protoplasm of the cells is finely granular, and often contains pigment particles. Not uncommonly they are observed to be undergoing

fatty degeneration, and occasionally are so altered as to be hardly recognisable, their protoplasm being replaced by large, highly refracting particles, named by Virchow "myelin droplets" (Fig 11. *b*), which may also be floating free in the sputum. Alveolar cells are found in connection with various diseases, and neither they nor the myelin droplets have any diagnostic value.

Elastic Fibres, when present, can nearly always be found in opaque portions picked out from the sputum as above directed.

Another method generally adopted is that introduced by the late Dr. Samuel Fenwick.[5] The whole of the sputum for twenty-four hours is collected and boiled for a few minutes in

FIG. 12.—ELASTIC FIBRES OBTAINED FROM SPUTUM AFTER A DOSE OF TUBERCULIN HAD BEEN ADMINISTERED.

a beaker, with an equal quantity of a solution of caustic soda, 20 grains to the ounce, and occasionally stirred with a glass rod, until it becomes diffluent. As soon as it is quite liquid, the mixture is thrown into a conical glass containing several times its bulk of water. In a short time a deposit will have settled, and portions of it may be removed with a pipette and placed under a microscope, when the elastic fibres will readily be recognised. The liquid may, if necessary, be centrifugalised. Care must be taken not to boil too long, or the fibres themselves will become swollen and ultimately dissolved.

The characteristics of these fibres (Fig. 12) are their sharpness of outline, indestructibility, dichotomous branching, and general contour marked by wide curves and loops. By these

appearances they may generally be distinguished, but sometimes threads of cotton or the mycelium of fungi may resemble them somewhat closely. Under these circumstances resort must be had to differential staining, elastic fibres taking a brownish-violet colour with acid solution of orcein, and resisting decolourisation on subsequent treatment with acid alcohol, whilst other matters are but faintly stained.

The late Sir Andrew Clark was the first to insist upon the important conclusions that may be derived from a minute examination of the elastic fibres. If they form complete *sheddings of the alveoli*, the morbid process is a very acute one, and destruction of the lung is proceeding rapidly. In more chronic conditions only small "tailed" pieces will be found, which have lost their elasticity. Fibres may sometimes be found which have been retained in the lungs for a long period (one to two years), and have become encrusted with lime salts (Fig. 13). Such incrustations disappear if a little dilute acid be run under the cover-glass.

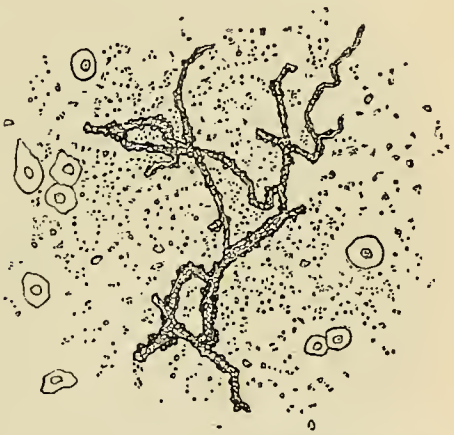


Fig. 13.—Elastic Tissue encrusted with Lime Salts.

Although met with chiefly and in largest numbers in the sputum of phthisis, elastic elements are also found in the expectoration from cases of pulmonary abscess, and occasionally in that of patients suffering from bronchiectasis or pneumonia. In cases of gangrene of the lung they are not met with as often as might be expected, being destroyed by the ferments formed in the process. Their presence in the sputum definitely indicates disintegration of lung tissue.

Occasionally elastic fibres, unlike those which we have been describing, are seen in straight bundles. If the individual fibres composing these are fine, they probably come from the bronchi or larynx; but if coarse, food-matter is probably their source, and this will be corroborated by the presence of small portions of muscular tissue adherent to them.

Larger fragments of lung tissue, in the form of dark grey masses, with tags and shreds, are occasionally met with under

similar circumstances to the above; and in cases of deep ulceration of the larynx portions of cartilage may be loosened and expectorated.

Curschmann's Spirals.—These bodies, first carefully described and figured by Curschmann,[6] have, as their name indicates, a peculiar spiral form. They may be recognised by the naked eye in the sputum as little whitish twisted bodies about an inch long and $\frac{1}{20}$ inch broad, and occasionally very much longer (Fig. 14). Under the microscope they are found to consist of a central highly refracting wavy thread, around which is coiled a network of fine fibres having a spiral arrangement, the whole being surrounded by mucus in which are embedded numerous leucocytes (Plate II.). In other cases the central thread is not visible, and the spiral appears to be composed of the enveloping coil only; in others, again, the central thread alone may be seen. Chemically, the central thread would seem to be composed of fibrin, the spiral meshwork encircling it being closely allied to mucin. The spirals were first observed in sputum coughed up during attacks of spasmodic asthma, and were considered to be diagnostic of that condition; but they have since been discovered in the expectoration of simple bronchitis, phthisis, pneumonia and œdema of the lungs, and probably indicate nothing more than a catarrh of the finest bronchial tubes.

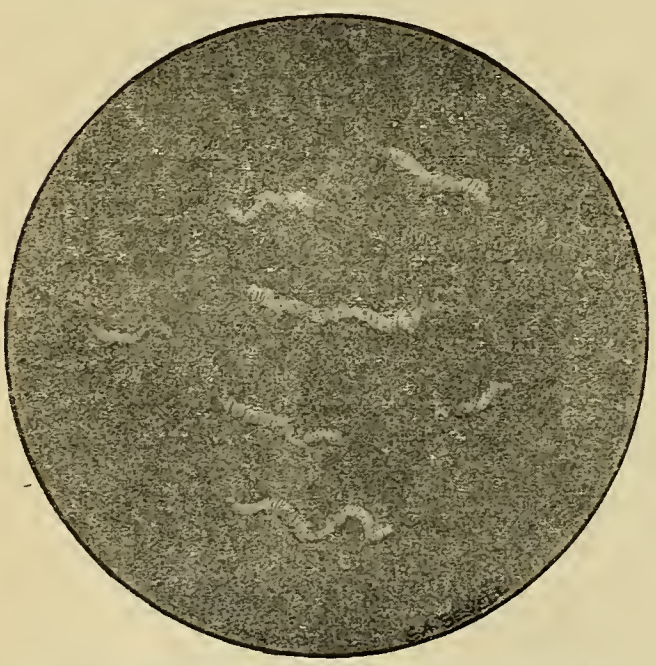


Fig. 14.—Showing Naked-Eye Appearance in the Sputum of Curschmann's Spirals.

Tonsillar Casts.—In cases of follicular tonsillitis casts of the tonsillar crypts are not infrequently found in the sputum. When teased out they are seen to consist of fine fibre-like bodies of varying length; more usually they occur in fragments.

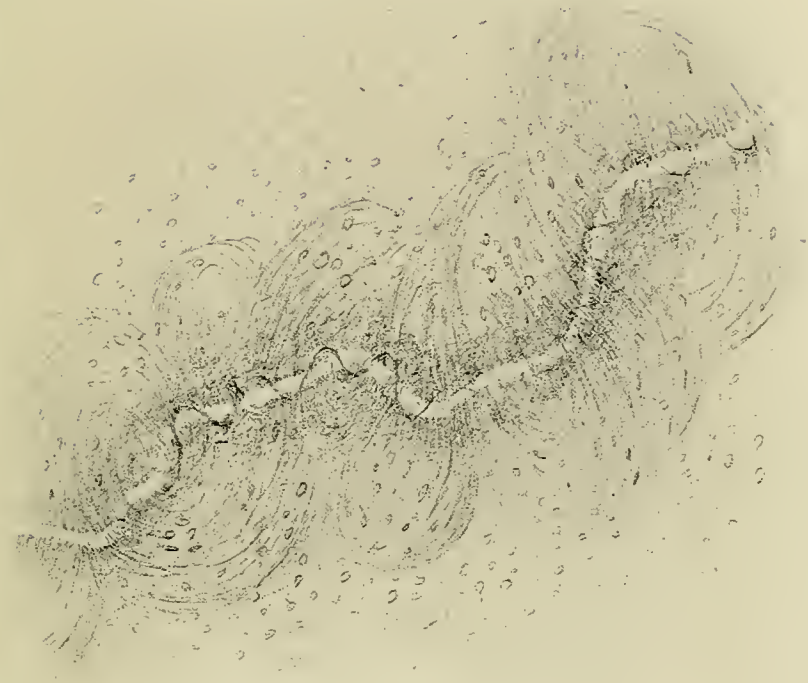
PLATE II

CURSCHMANN'S SPIRALS

The lower drawing, which may be compared with Fig. 14, illustrates a spiral under a low degree of magnification. The coiled appearance of the spiral is well represented. (After Curschmann.)

In the upper drawing a spiral is seen under a higher power. In the centre is a highly refractive thread, around which a delicate festoon is coiled, the whole being surrounded by mucus, in which many cells are embedded. (From a drawing by Dr. W. C. Fowler. × 96.)

PLATE II



CURSCHMANN'S SPIRALS.

To face p. 74.

Crystals.—Crystals of various forms are occasionally met with in sputum, the best known being the "Charcot-Leyden crystals" (Fig. 15). These occur as colourless pointed octahedra, and are identical with those sometimes seen in the blood in leukæmia. Their chemical nature is uncertain. Clinically their significance is small, for though found, often in considerable numbers, in the sputum after an asthmatic paroxysm, they, like the spirals, may also occur in connection with phthisis, bronchial catarrh and plastic bronchitis, as well as other diseases. They would seem to arise from the disintegration of the cellular elements in the sputum.

Cholesterin crystals are principally associated with collec-

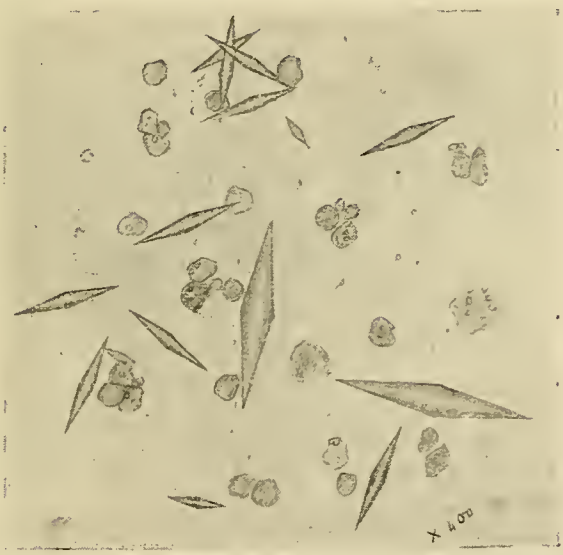


FIG. 15.—CHARCOT-LEYDEN CRYSTALS, FROM A CASE OF ASTHMA. (From a drawing by Dr. W. C. Fowler : × 400.)

tions of pus in pulmonary abscess, empyema and fœtid bronchiectasis, but are not confined to these conditions.

Crystals of fatty acid appear as long slender needles, often formed into rosettes. They are very common in the sputa of bronchiectasis and gangrene of the lungs, but in small numbers occur in any muco-purulent expectoration, especially if it has been kept in a warm place.

Crystals of hæmatoidin are brownish-yellow or red in colour, and take the form of rhombic prisms (Fig. 11, *e*). They indicate that blood has been retained for some time in the air-passages, and are numerous after an hæmoptysis.

Crystals of tyrosin, oxalate of lime and triple phosphates, occasionally occur, but their presence is of no significance.

Micro-organisms.—In addition to the bodies which we have so far described in the sputum, numerous micro-organisms are to be found.

Some of these, such as the tubercle bacillus, the streptococci, the pneumococcus, varieties of the streptothrix group, and many others, are pathogenic in nature, and play a fundamental rôle in the ætiology of pulmonary disease. It is not our purpose in this chapter to describe these organisms. To many we shall refer again, when the diseases originated by them are discussed, and for a more detailed description we must direct the reader to special works on bacteriology.

Others are saprophytic in nature, or if pathogenic, very feebly so. We may, for instance, find in the expectoration the long bacillary threads of a variety of leptothrix, or little packets of cocci forming cubes of eight produced by certain strains of sarcinæ. Neither possess any pathological significance, though a hasty observer might mistake the former for leashes of elastic tissue. Varieties of saprophytic moulds may also be encountered in the sputum, and one has been described by the late Mr. Coppen Jones[7] which occurs not uncommonly in phthisis, in which disease also the tetrads, or groups of four cocci, formed by the Micrococcus tetragenus, are frequently to be found. Occasionally the thrush fungus, Oïdium albicans, embedded as a rule in masses of epithelial débris, is present in the expectoration.

Adventitious Matters.—The sputum sometimes contains other substances than those already referred to. In a few cases these bodies are of much diagnostic importance; for instance, the fragments of new growth sometimes coughed up in the course of malignant disease of the lung, the hooklets of hydatids, the ova of the lung fluke, *Paragonimus Ringeri*, in endemic hæmoptysis, and the hairs occasionally yielded by mediastinal dermoid cysts. Of pathological interest also are the white cretaceous masses coughed up by certain phthisical patients.

It must not be forgotten that the sputum may contain matters which are purely extraneous, such as grape-skins, nutshells, orange-pips, linen, cotton, silk or woollen fibres, particles of animal and vegetable tissues, starch granules, fat, and so forth. These are obviously accidental, and have only found their way into the spittoon by chance; but, unless their

nature be recognised, they are likely to be mistaken for some of the pathological constituents of the expectoration, which we have already described.

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CHAPTER V

DEFORMITIES AND DISEASES OF THE CHEST WALLS

THE shape of the chest is nicely conformed, as a rule, to the condition of the lungs. Thus we have the small, long thorax with oblique approximated ribs associated with small lungs; the large, expanded, barrel-shaped chest with emphysematous lungs; and the local flattening or enlargement met with in certain pulmonary or pleuritic diseases. To these we shall have occasion to refer again. We may now consider certain alterations in the shape of the chest which deserve special attention.

1. **The Pigeon Breast.**—On the surface of the chest in the inframammary region of each side there exists an area, having the fifth space in the nipple line for its centre, which is unsupported by muscles. This space occupies the interval between the insertions of the pectoralis, the serratus, and the rectus muscles, and in any obstruction to the entry of air into the lungs, the atmospheric pressure is here least supported by muscular action. Hence, in young children in whom the ribs and cartilages readily yield in any condition of general dyspnœa, this part of the chest on each side becomes depressed and the sternum tilted outwards. Repeated bronchial attacks thus give rise to the prominent sternum and depressed inferior and lateral thorax which persist and constitute the pigeon breast, a deformity which is intensified by the drawing in of the lower chest by the action of the diaphragm along its zone of attachment.

2. In the **Rickety Thorax** a more or less deep groove corresponds on each side with the junction of the ribs and their cartilages, there being here a knuckling in, so to speak, of the ribs, causing the sternum to come forward with undue prominence. This is due to atmospheric pressure depressing the

softened and unresisting portions of the ribs and their adjacent cartilages.

3. **The Alar Chest.**—It has been known since the time of Aretæus,* and possibly was recognised even before, that in patients suffering from phthisis, or merely predisposed to this disease, the chest is often small and narrow. As a consequence there is insufficient breadth of support for the scapulæ, the superior angles of which are thrown forward and inward by the weight of the arms, and the inferior angle tilted outwards. The effect upon a thin subject, when viewed from behind, is thus to produce a winged appearance, to describe which the terms "alar" or "pterygoid" have been employed.

Another deviation from the normal in the shape of the chest is the *cup-like hollow*, which is from time to time seen over the lower portion of the sternum and the adjacent cartilages. This is sometimes produced by occupations, such as that of a shoemaker, in which persistent pressure is made upon the lower sternal region. The deformity also occurs in a marked degree when no history of pressure can be obtained, and may then be attributed to obstructed respiration during childhood.[2]

Lastly, we may mention a deformity due to a *deficiency of the clavicular portion of the pectoralis major muscle* on one side, of which we have seen some examples. A remarkable flattening, or rather hollowing, below the clavicle is thus occasioned, suggestive of grave internal lesion. The true cause of the depression is, however, at once rendered obvious by making the patient grasp the back of a chair with both hands and attempt to lift it, when the existing muscular fibres start into action, and the defect is declared.

Let us now turn to the diseases of the chest walls, which are of importance chiefly from the frequency with which they simulate more deeply-seated lesions.

Pleurodynia (πλευρά, the side, ὀδύνη, pain).—This may be due to *rheumatism*, *myalgia*, or *intercostal neuralgia*. The symptoms are very similar in all these conditions, but more particularly in the two first-named, and there can be no doubt

* Aretæus[1] (*circa* A.D. 50) thus describes the chest of phthisis: "... the whole shoulder-blades apparent like the wings of birds. ... The habits most prone to the disease are the slender; those in which the scapulæ protrude like folding-doors, or like wings; in those which have prominent throats; and those which are pale and have narrow chests."

that aponeurotic rheumatism and myalgia are conditions frequently associated and difficult to distinguish. Pleurodynia commonly begins quite suddenly with severe pain of a tingling or burning character much aggravated by respiratory movements, especially those of deep inspiration or tussive expiration. The pain may be situated at any part of the chest, but a common seat is over the insertion of the pectoral muscle in front, or at the margin or sides of the chest where the recti and serrati muscles are attached. When the pain is lateral, it is limited to one side. The breathing of the patient is hampered, and may even be greatly distressed by the severity of the pain.

Aponeurotic Rheumatism is not common before thirty, although it may occur in young people of distinctly rheumatic diathesis. It is frequently consequent upon direct exposure to cold, especially in persons coming from hot rooms and when overheated by exertion. Middle-aged and elderly people, who are the subjects of gout and allied rheumatic affections, such as lumbago and sciatica, are specially liable to this affection.

Myalgia, except in so far as muscular tenderness is inevitably more or less associated with the preceding condition, is generally traceable to strain. It sometimes occurs in whooping-cough, and very commonly in the course of phthisis. Unquestionably the disease has in most instances a definite pathology in the rupture of some minute muscular fibres.

Intercostal Neuralgia is a more definite ailment than either of the above, and occurs more especially in females, at two periods of life, and in two forms. (1) It is very common in young women in the form of inframammary neuralgia, in which the pain is referred to the surface of the chest below the left breast. This affection is most generally associated with anæmia attended with leucorrhœa, amenorrhœa or other menstrual disturbance. (2) It also occurs in persons of middle or advanced middle life and, again, is more common in females. In this form the pain is of a very severe stinging or burning character along the course of the peripheral distribution of one or more of the intercostal nerves. It may last for days or even weeks, and then be followed by the appearance of the characteristic eruption of herpes zoster, after which the pain is soon mitigated, or subsides altogether. This disease appears to be commonly associated with the rheumatic or gouty diathesis. Although most common in females of

middle age, it is by no means limited to them, and may sometimes be observed in quite young people. The pain attendant upon herpes zoster in the young, however, is not often severe, and rarely precedes the appearance of the eruption by any considerable time.

Diagnosis.—The distinctive signs of pleurodynia, when carefully considered, are quite sufficient for diagnosis:

1. The disease *per se* does not raise the temperature, in which fact we have one very important element in the diagnosis from pleurisy.

2. There is usually decided tenderness on pressure over the seat of pain. In intercostal neuralgia this tenderness is very superficial and is best elicited by gently pinching up the skin. The tenderness may be observed, too, not only over the point most complained of, but also in the lateral spinal region corresponding with the distribution of the posterior cutaneous branches of the affected nerves. In cases in which the pain is chiefly situated below the left breast, the patient usually presents other signs of anæmia. In myalgia the pain is markedly elicited by bringing the muscles affected into action, asking the patient, for example, to grasp the back of a chair, thus bringing the pectoral muscles into action, or to raise himself in bed or turn on one side so as to involve other muscles. In aponeurotic rheumatism and in myalgia there is also tenderness on firm pressure over the muscles in the intercostal spaces.

3. The respiratory movements are restrained by the pain, and respiration on the affected side is correspondingly weak; but it is of vesicular character, and unattended with any friction sounds. One may sometimes hear, however, muscular vibratile sounds, due to the voluntary restraint exercised upon this side by the patient. The percussion resonance is unaltered.

Pleurisy and pericarditis are the two conditions most simulated by pleurodynia. In phthisical subjects, where there is perhaps already existing pyrexia, it is often difficult to be sure that there is not some dry pleurisy, but here the diagnosis is not very important. Aponeurotic rheumatism of the front of the chest is distinguished from pericarditis by the absence of the physical signs proper to that disease.

Treatment.—Intercostal neuralgia in young anæmic subjects

requires general treatment by iron, quinine, arsenic, and fresh air, with generous diet; also local treatment by soothing applications, such as belladonna plasters, or chloroform liniment (3 parts) with belladonna liniment (1 part), sprinkled on lint or flannel backed by oiled silk. Menthol, dissolved in liniment of chloroform (ʒi. in ℥i.), may be applied in a similar manner, or the Linimentum Aconiti Compositum containing equal parts of the liniments of aconite, belladonna, and chloroform. Aconite and belladonna may also be efficaciously applied in the form of the Chloroformum aconiti, or Chloroformum belladonnæ of Messrs. Squire, the remedy being painted with a camel's-hair brush over the painful part. Another preparation which sometimes gives relief when painted on the skin is "Kasemol," which is believed to contain menthol, methyl salicylate, the oils of mustard and sassafras and the essential oil of camphor. In the herpes zoster form of intercostal neuralgia, before the eruption appears, the same remedies may be used; afterwards the local application must be used with caution. In this latter variety of neuralgia, the pain is often very severe and intractable, and subcutaneous injections of morphia and atropine may be required. Local depletion by leeches will always give temporary relief, but in the anæmic form of neuralgia this method of treatment is not to be advised. The myalgic forms of pleurodynia are best treated by keeping the affected side of the chest at rest by strapping, as for dry pleurisy. Aponeurotic rheumatism may often at once be cured by the application of a well-made mustard poultice kept on for twenty minutes. In the case of any rheumatic diathesis, general remedies, especially iodide of potassium with quinine and salicylates or aspirin, will be required.

Periostitis and Perichondritis of Sternum and Ribs.—This is a disease which comes under the notice of the physician not very infrequently, and which sometimes gives rise to perplexity in diagnosis. It is generally the result of syphilis, typhoid fever or tubercle, and is occasionally met with in the course of pulmonary phthisis.

When due to tubercle, the tuberculous process may start as a small area of central caries in the middle of the rib (Fig. 16), and then spread through the bone to the periosteum; or it may commence near the surface, and attack the superficial layer of bone and the periosteum simultaneously. In which-

ever way it starts it eventually forms an abscess, which as a rule points forwards; but if the disease has commenced on the inner aspect of the rib or sternum, a large collection of pus may form here, separating the pleura from the ribs, compressing the lung, and simulating most closely an empyema. These cases are, happily, rare, and as a rule the formation of a small swelling over the sternum or an adjacent rib, accompanied by but little pain, is the first sign which attracts the patient's attention and causes him to seek advice. If treated at once surgically by evacuation of the pus and removal of any dead bone, the disease is generally cured; but if left untreated,

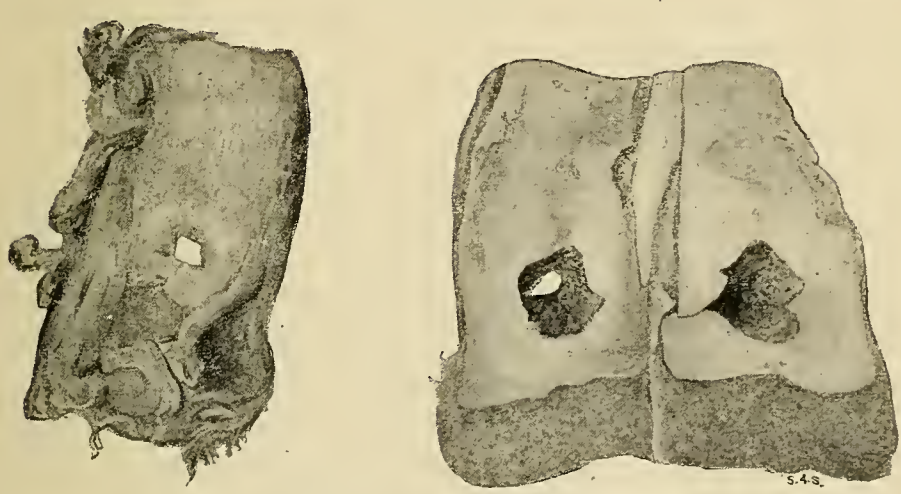

FIG. 16.—TUBERCULOUS OSTEO-MYELITIS OF RIB.

The specimen shows the outer surface of the fourth right rib and cartilage, and also the appearances seen when a section was made and the parts opened outwards. A central area of disease is visible, the size of a pea, which communicated during life with a costal abscess by the small hole seen in the figure. Removed by operation from a man suffering from chronic pulmonary tuberculosis. (From the Brompton Hospital Museum.)

the pus may burrow and cause extensive lesions. We have seen complete necrosis of the sternum thus produced.

In the case of typhoid fever, the inflammation, whether in connection with the sternum or a rib, is generally produced by the direct action of the typhoid bacillus, though sometimes it is the result of secondary infection by the pyogenic micro-organisms.[3] The symptoms resemble those of the tuberculous cases, although they may terminate in spontaneous resolution. In a case of this kind seen by one of us some time ago, the symptoms, local prominence over the first two ribs, obscured breath-sounds, some venous engorgement, and great tender-

ness, were traceable to an attack of typhoid fever four or five months previously, and suggested at first the diagnosis of a local abscess or empyema. The lady, however, ultimately, though slowly, recovered, without operation, under repeated and prolonged change of air and other remedies.

In syphilis, periosteal nodes over the sternum (especially its upper portion) and over the ribs are not uncommon; more rarely gummata breaking down into ulcerating sores may be observed. Tubercle or malignant disease may be suspected, but it should be a rule in any doubtful case to administer a course of antisyphilitic treatment. Occasionally pulsation may be conveyed to a softened gumma, and suggest the diagnosis of aneurism.

Fascial Creaking.—Before concluding this chapter, we must mention a very curious physical sign, the so-called fascial creaking which may be heard, especially over the supraspinous and scapular regions. This phenomenon is attended with a certain amount of pain. We have met with cases of this kind which have been treated with great perseverance for many weeks by blisters and iodine counter-irritants, under the impression that the patients were suffering from chronic pleurisy. The condition is, however, so far as we have been able to observe, a permanent one, and is only important in diagnosis. If the attention be once awakened to the possibility of the sounds being extrathoracic, the diagnosis can be confirmed by making the patient stop breathing and by listening whilst the shoulder is shifted about, when the creaking will still be heard.

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CHAPTER VI

DISEASES OF THE PLEURA

Pleurisy.

THE pleura on each side of the chest is a closed serous cavity or sac intimately applied to, and in organic union by its outer surface with the lung and the costal parietes. The internal surface of the sac is lined with endothelium, the costal and parietal portions being in close contact, lubricated merely by some moist serous secretion. This contact or apposition is maintained by atmospheric pressure bearing upon the interior of the lung and the exterior of the chest wall, which more than counterbalances the constant tendency of the two surfaces of the sac to spring apart from the opposite elastic tractions of the lung and thoracic wall.

The pleura is well provided with bloodvessels, which are derived from the bronchial, internal mammary and intercostal arteries.

The lymphatics consist of two series, the one forming a network beneath the surface endothelium, the other situated in the cellular tissue subjacent to the pleura. Both series communicate freely with each other. In the visceral portion of the pleura the lymphatics communicate, as we have indicated elsewhere (see p. 18), with the pulmonary lymphatics, and both are drained by the superficial collecting trunks into the bronchial glands at the hilum of the lung. The parietal pleura, so far as its costal portion is concerned, is drained by the deep intercostal lymphatics, and thus by the internal mammary vessels and glands; the lymphatics of the mediastinal pleura terminate in the posterior mediastinal glands. The communication of the superficial and deep intercostal lymphatics, and the connection of the former with the lymphatics of the chest wall, explains, as Poirier

and Cunéo[1] point out, the affection of the axillary glands which is sometimes observed in deep-seated thoracic disease. The important connection existing between the lymphatics of the pleural and peritoneal surfaces of the diaphragm, and also between the lymphatics of the liver and those of the diaphragmatic pleura, has already been referred to (see p. 19).

When we regard the position and connections of the pleura, its extensive endothelial surface, the conditions of negative pressure in which that surface is ever maintained, and its richness in lymphatics, we cannot wonder that it should frequently become the seat of disease, nor that it should be one of the chosen sites for the manifestation of lesions resulting from blood infection. Few are the autopsies after adult age in which one fails to find some imperfections in the pleura; few are the cases of septic or pyæmic poisoning in which this membrane is not actively involved.

Pleurisy, as the physicians of the early Roman Empire knew full well, is an inflammation of the pleural membrane. "Under the ribs, the spine, and the internal part of the thorax, as far as the clavicles"—so writes Aretæus[2]—"there is stretched a thin, strong membrane, adhering to the bone. . . . When inflammation occurs in it . . . the affection is named pleurisy." Clinically, however, cases differ much among themselves, and various forms of the disease are recognised. A classification based upon bacteriology alone would not give an adequate presentment of the disease in its varying characteristics. It has not as yet been shown that in all cases a micro-organism is responsible for it, although no doubt it is so in the great majority of instances. Again, the same microbes, for example, the pneumococcus or the tubercle bacillus, will produce at different times and in varying circumstances quite different forms of pleurisy. We shall, therefore, under the common heading "Inflammation of the Pleura," describe the ætiology and characters of the following groups, viz.: (*a*) Plastic or dry pleurisy; (*b*) Pleurisy with effusion, whether sero-fibrinous, hæmorrhagic or purulent.

1. **Fibrinous, Plastic, or Dry Pleurisy.**—This variety of pleurisy consists in an inflammation of the membrane, leading to the exudation of lymph, usually over a limited area of its surface. Beyond this stage the inflammatory process does not pass, and after a time the lymph is absorbed, or

adhesions form, binding the surfaces to each other. The disease may be either primary—occurring, that is to say, in the absence of any evident lesion of the lung—or secondary to the spread of inflammation (whether tuberculous or otherwise) from the lung or chest wall, or to some general blood infection. True primary cases are rare, many of those which at first seem to be of this nature proving eventually to be the result of tuberculous disease of the lung which has not as yet given rise to symptoms.

Pathology.—The pathology of the disease may be briefly described as consisting in the first place of hyperæmia of the pleura; within a very few hours the normal glistening appearance of the surface is lost; it becomes cloudy, as though breathed upon, and gradually covered with a layer of exuded lymph. Both the costal and visceral layers are thus affected, and the result of the movement of the two surfaces upon each other is to roughen them, causing the effused lymph to present numerous little elevations and pittings, like the fretted surface of a lake.

At this point the disease may come to a standstill, and the exuded lymph be gradually again absorbed. The pleura in this case once more becomes smooth, but the site of the old lesion is generally revealed by a thickening and opacity of the membrane, and sometimes by a slight radiated puckering, due to the formation of a thin layer of fibrous tissue. More often the lymph exuded is not simply absorbed, but vascular loops extend into it from the pleural vessels on either side, which meet and inosculate, giving rise to the formation of permanent adhesions. As a rule, these are not of great density, but where there is much shrinking of the lung, as in many cases of phthisis, they become, as we have shown elsewhere,[3] stretched and filled with fluid, and present a gelatinous aspect (Plate XXII., *Frontispiece*). Organisation slowly follows, and leads to the formation of firm and dense adhesions, which measure not uncommonly at the apex of the lung $\frac{1}{3}$ inch or more in thickness.

On rare occasions, without previous disease of the lung, tubercle attacks the pleura, and gives rise to a somewhat similar condition, termed by the late Sir William Osler[4] "chronic adhesive tubercular pleurisy." In this disease the pleura over the whole lung becomes, by the gradual develop-

ment of tubercles, greatly thickened, measuring, perhaps, ½ to 1 inch across. On section, the tissue is found to consist of fibroid material, with small areas of interspersed caseation. The condition is a very rare one, and we remember to have seen only one example, which was probably of this nature.

Symptoms.—The symptoms of acute dry pleurisy set in as a rule somewhat suddenly. The patient complains of cough and pain in the side, increased on deep breathing. The temperature is found to be somewhat raised, reaching perhaps 100° or 101°. The percussion note is natural, and the breath-sounds vesicular, though weak and uneven. The diagnosis depends upon the detection of a friction sound, the characteristics of which have been already described (see p. 60). This is heard over the seat of pain, generally in the lower posterior or lateral regions of the chest. The disease generally lasts but a few days, when the temperature falls and the friction sound disappears. If the pleurisy be secondary to some other disease, such as pneumonia, pulmonary infarct, phthisis, or gangrene, its symptoms will merge into those of the primary disease, but, as a rule, some pain in the side (in pneumonia often of great severity) will be complained of, and a friction sound is generally detected.

Diaphragmatic Pleurisy.—When the pleurisy is restricted to the surface of the diaphragm, the symptoms present certain peculiarities. The onset of the disease is marked by severe pain in the lower portion of the chest corresponding to the insertion of the diaphragm. The pain, as Gueneau de Mussy pointed out, is especially felt along the tenth rib, extending from the anterior extremity to the sternum and ensiform cartilage. Sometimes it radiates to the back and shoulders. The hypochondrium is tender on pressure. Respiration is hurried, but the diaphragm on the affected side is kept motionless, since every contraction aggravates the pain. The patient is feverish, and looks ill and anxious, but a careful physical examination at first reveals nothing to account for the somewhat alarming symptoms, which may after a short time decline, and recovery take place. If effusion follows, the characteristic signs will suggest the true nature of the disease. It is easy for the physical signs of diaphragmatic pleurisy to be overlooked, and in purulent cases the diagnosis may be made only on a sudden discharge of pus through the lung.

We must add that, though the disease generally manifests itself by severe symptoms, it may be masked by the other conditions present. We have seen a case of phthisis in which the autopsy revealed a tuberculous diaphragmatic empyema containing half a pint of pus, although during life there had been no symptoms to suggest the presence of such a complication.

For the points which should be attended to in forming a diagnosis between pleurisy, intercostal neuralgia and rheumatism we must refer the reader to what we have already said upon the subject (see p. 81).

Treatment.—In many cases of dry pleurisy immediate relief is given by firmly strapping the affected side, so as to restrain its movement, as first recommended by the late Dr. Roberts. A useful clinical test as to the amount of relief which may be thus expected is obtained by observing the effect of steady and firm pressure by the hand upon the affected side. In some cases strapping cannot be borne, either on account of its increasing the pain or from the fact that, as may happen in phthisis, the pleurisy has supervened on the side most available for respiration. Under such circumstances the application of a small blister, with or without a hot linseed poultice superposed, will give relief or, if the pain be severe, three or four leeches may be applied. In cases associated with pneumonia the latter remedy is of great value. An opiate or subcutaneous injection of morphia may be employed if necessary, simultaneously with one or other of the above remedies.

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CHAPTER VII

SERO-FIBRINOUS AND HÆMORRHAGIC PLEURISY

Acute Sero-Fibrinous Pleurisy.

SERO-FIBRINOUS pleurisy does not differ in its early stages from the acute dry pleurisy just considered. The pleural surfaces are inflamed and lymph is exuded; but whereas in dry pleurisy this constitutes the whole disease, in the variety now under consideration there is, in addition, an effusion of fluid, for the most part serous in nature, and sometimes amounting to many pints in quantity.

Ætiology.—This disease may occur in the course of other maladies, such as phthisis, rheumatic fever or pneumonia. In these cases the pleurisy would seem to depend upon the agency of the micro-organism which has produced the original disease. When occurring in association with malignant disease of the lung, effusions into the pleura are due perhaps as often to obstruction of bloodvessels and lymphatics as to definite inflammation of the serous membrane. Pleurisy, in common with other serous inflammations, also occurs in the course of acute and chronic Bright's disease, but the effusion into the pleura met with in this disease is more frequently of a passive nature.

Concerning the *primary cases*, in which the pleurisy apparently arises *de novo*, and which constitute the bulk of all sero-fibrinous effusions, it was held until recently that exposure to cold or draught was the chief factor in their causation, whence the term *pleuritis a frigore*. This view we now know to be incorrect, and no doubt the great majority of such cases are really tuberculous in nature. In certain instances, however, a pleurisy which to the naked eye appears serous in character, though the microscope will, as a rule, show excess of polymorphonuclear leucocytes in the fluid, is due to the

presence of the pneumococcus or, as in the recent influenza epidemic, to the streptococcus, although in most cases these give rise to a suppurative pleurisy.

The evidence upon which is based our belief as to the tuberculous nature of most primary serous effusions may be summarised as follows:

(1) If we trace the after-history of these cases, we see that in many of them apparent recovery is shortly followed by the development of phthisis; thus, Dr. Hedges,[1] following up 130 cases from St. Bartholomew's Hospital, found that within six years or less "43 per cent. had either died of phthisis or other tubercular lesion, or presented signs of the former disease." Very similar results were obtained by Dr. Barrs,[2] and more recently by two Swedish observers, Drs. Allard and Köster.[3] The conclusion, therefore, that the pleurisy in these cases was in reality tuberculous from the first is strongly suggested, and is further strengthened by the fact that in certain rare cases, such as those recorded by Kelsch and Vaillard,[4] in which the patients died, tubercles have been found in the serous membrane.

(2) An examination of the fluid by staining methods affords confirmatory evidence. If the fluid withdrawn by aspiration be examined without special preparation, bacilli are as a rule not found. If, however, we follow Dr. Jousset's[5] method, and allow the fluid to clot, then digest the clot with artificial gastric juice, and finally stain films made from the centrifugalised liquid, tubercle bacilli can often be detected. Jousset himself claimed to have demonstrated them in seventeen successive cases of primary serous pleurisies.

(3) Inoculation experiments tell a similar tale, provided large enough quantities of fluid be injected. Netter[6] found that in eight cases out of twenty of primary sero-fibrinous pleurisy he was able to induce tuberculosis in guinea-pigs by injecting into the peritoneal cavity 1 to 1½ c.c. of the fluid withdrawn by aspiration. Eichhorst,[7] using a larger quantity (15 c.c.), produced tuberculosis in 65 per cent. of his cases; while Le Damany,[8] by injecting 10 to 50 c.c., and in one case even 300 c.c., was successful in forty-seven out of fifty-five cases, or 85 per cent.

These statements are not gainsaid, and we are driven, therefore, to the conclusion that by far the greater number

of cases of primary sero-fibrinous pleurisy are in reality tuberculous in nature—a fact which must greatly influence our after-treatment of the malady.

Morbid Anatomy—Nature of the Fluid.—As we have seen, the morbid changes are at first similar to those observed in the plastic or dry variety of the disease. Hyperæmia first occurs, to be followed shortly by roughening of the surface and the deposition of lymph. Effusion, however, then takes place, which, as it increases, separates the surfaces more or less widely from each other. The fluid thus effused is clear, yellowish in colour, from the presence of serum-lutein, and alkaline in reaction. It is highly albuminous, and on standing undergoes spontaneous coagulation; but the amount of fibrin which separates out is never great. On microscopical examination, leucocytes are seen, also a few large cells, derived from the endothelium lining the pleural surface, together with a fair number of red corpuscles. The leucocytes, as Widal and Ravaut[9] were the first to show, are mostly small mononuclears, and so constant is their presence in great excess in cases of tuberculous pleurisy,[10] except in its earliest stage, that a cytological examination will be found of value when considering the diagnosis of any case. It should be stated, however, that in cases of malignant pleurisy a similar excess of small mononuclears is often to be observed.*

According to Professor Halliburton,[11] whose researches have been confirmed by other observers, the average chemical composition of the fluid may be given as follows, the corresponding figures for passive effusions (chronic renal and heart disease) being quoted for comparison:

	Specific Gravity.	Total Proteids per Cent.	Fibrin.	Serum Globulin.	Serum Albumin.
Acute pleurisy ...	1021	4·5903	0·0473	2·0007	2·2112
Hydrothorax ...	1014	1·7748	0·0086	0·6138	1·1558

* If the fluid, after withdrawal from the chest, cannot be at once examined by the pathologist, it is well to add one-quarter its volume of a citrate solution of the following composition:

Sodium citrate	1·5 grms.
Sodium chloride	0·85 grms.
Distilled water	100 c.c.

In this way clotting is prevented and the cells are preserved.

From these figures it will be seen that in acute sero-fibrinous pleurisy the fluid is of higher specific gravity and richer in proteids than in non-inflammatory effusions, whilst the yield of fibrin is also greater, leading to more rapid coagulation of the fluid. The proportion of serum albumin to serum globulin is variable, and possesses no clinical importance.

In cases of chronic pleurisy, according to the analysis of Méhu,[12] the fluid would seem more closely to resemble that of hydrothorax.

Symptomatology.—The following description of the disease will apply chiefly to the common primary variety, the *pleuritis a frigore* of older writers; but the symptoms and physical signs of cases complicating other diseases do not differ in any essential particular.

The symptoms and signs of sero-fibrinous pleurisy are in accordance with its pathology, and are divided into three stages: (1) the stage of hyperæmia and commencing exudation; (2) the stage of effusion; (3) the stage of absorption and convalescence.

Pains in the side and shivering are the two symptoms which usher in the attack, and either may precede the other by a few hours. The pain is that of an acute "stitch" in the side, usually felt in the lower axillary or inframammary region, but sometimes referred, and especially in children, to a much lower point in the abdominal wall to which the terminal cutaneous twigs of the affected intercostal nerves are distributed. The pain interferes with the respiratory movements, which are restrained and shallow, the patient inclining to the affected side so as to lessen its movement. The shivering is of variable severity, sometimes very sharp and decided—a true rigor—at other times amounting only to recurring chills. It is stated that the rigors of pleurisy are repeated, whilst in pneumonia one severe shivering occurs at the commencement of the disease; but in neither case is this statement more than generally speaking correct. There is occasional dry, interrupted cough. The temperature as a rule rises rapidly to about 102° or 103° (see Figs. 17 and 18), the face is anxious and pale, the pulse small and moderately frequent. The fever has never the marked character of that of pneumonia, and the flushed cheek and burning skin so characteristic of the latter disease are rarely present. In a word,

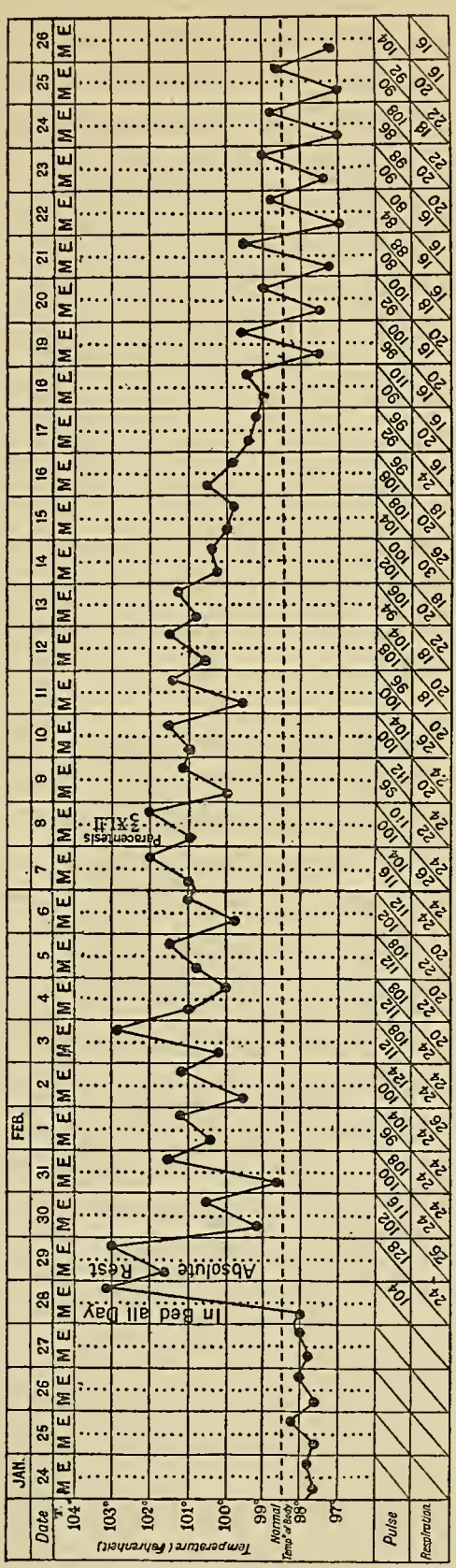

Fig. 17.—Chart showing the Temperature Record of S. P. (Male), æt. 30, Clerk, who, while an In-Patient at the Brompton Hospital, suffering from Chronic Phthisis, developed, on January 28, Acute Left Pleurisy with Effusion, which necessitated Paracentesis.

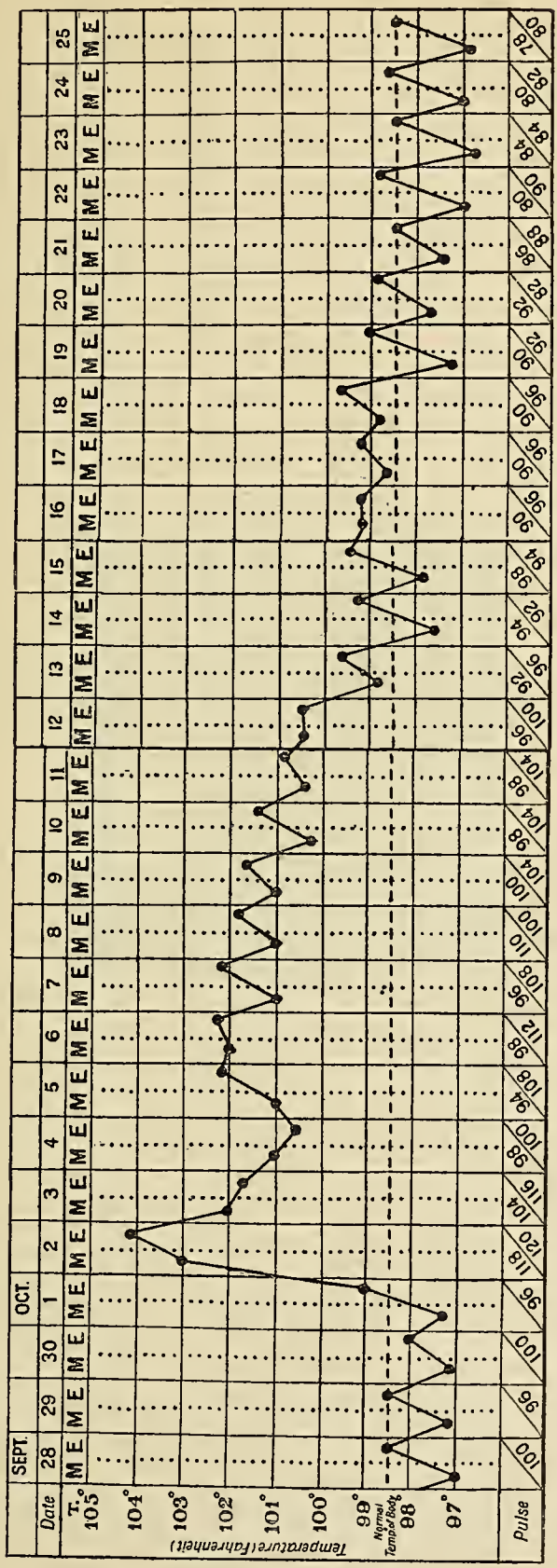

FIG. 18.—CHART SHOWING THE TEMPERATURE RECORD OF C. M. (MALE), ÆT. 21, WHO, WHILE A PATIENT AT THE BROMPTON HOSPITAL SANATORIUM, FRIMLEY, SUFFERING FROM PULMONARY TUBERCULOSIS, DEVELOPED, ON OCTOBER 1, ACUTE PLEURISY WITH EFFUSION OF THE LEFT SIDE.

the patient with acute pleurisy is not so ill as one with pneumonia, although he may be in more suffering.

In the first stage of the disease there is no percussion dulness. On listening to the chest, the respiratory murmur will be found to be uneven and partially suppressed on the affected side, and a more or less distinct friction sound, usually of a grating character, will be heard. This is most distinct at the end of inspiration and the beginning of expiration, and is best heard over the seat of pain, usually in the mammary or inframammary region, or over the base of the lung. In left-sided cases friction of cardiac rhythm—the so-called pleuro-pericardial friction (see p. 60)—may be heard over the region of the heart. Friction fremitus is occasionally to be felt. In some cases, however, which present all the symptoms and febrile phenomena characteristic of pleurisy, no friction sound can be heard. This may be explained by the manner in which the affected side of the chest is held fixed by the patient, or by the pleuritic exudation commencing on the diaphragmatic or mediastinal aspect of the pleura, and thus being beyond the reach of appreciation by the stethoscope.

Within a short time—it may be but a few hours—fluid commences to be effused, and dulness may be detected at the extreme base posteriorly, gradually extending upwards towards the apex. With the occurrence of effusion the pain becomes less, the breathing easier and less catching, although quicker than natural. The movements of the affected side are notably lessened, and the respiratory sounds more or less effaced, in marked contrast to their exaggerated intensity on the sound side. In moderate degrees of effusion, and especially in the early stages before adhesions have had time to form, the dulness varies slightly with the position of the patient; when lying down, for instance, the resonance may be good to just below the nipple, whereas on sitting up there is dulness up to the nipple, and it may be higher. As the fluid increases, the respiratory murmur soon becomes diminished or inaudible over the lower portion of the dull area, and friction can here no longer be detected. Too much importance must not, however, be attached to auscultatory evidence in the diagnosis of pleuritic effusion. As the upper limits of dulness are approached, distant tubular breathing

may be heard in the scapular region, whilst friction sound of a moister character is often audible, especially in front. With the advance of the effusion the breathing becomes increasingly distressed, and may amount even to orthnopnœa.

The signs of pleuritic effusion may be divided into three groups:

(*A*) *The cardinal signs of pleuritic effusion,* the presence of which are alone essential for diagnosis—viz. (1) percussion dulness; (2) displacement of the heart; (3) annulled vocal fremitus; (4) diminished and altered or absent breath-sound. These signs are common to all varieties of effusion, whether serous, purulent or hæmorrhagic.

(*B*) *Subordinate or supplementary signs*—viz. (1) alteration

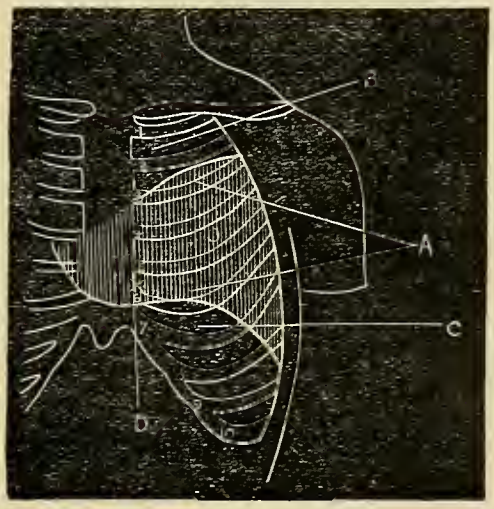

FIG. 19.—PERCUSSION SIGNS IN CASE OF MODERATE EFFUSION.

A, area of complete dulness ("flatness"); B, area of tympanitic (skodaic) resonance; C, inferior curved line of tympanitic (stomach) resonance.

in shape of chest and increase in semi-circumference; (2) intercostal bulging, elasticity, or fluctuation; (3) skodaic resonance; (4) altered voice-sound; (5) displacement of abdominal viscera; (6) signs in the other lung; and (7) cardiac displacement bruits. These signs are none of them essential for diagnosis, and any or all of them may be wanting.

(*C*) *Signs indicative of the nature of the fluid*—viz. (1) temperature signs; (2) other pyrexial or septic phenomena; (3) pectoriloquie aphonique (Baccelli); (4) the result of exploratory puncture.

1. The *dulness* of effusion is absolute and toneless. It is

distinguished by our American confrères from more ordinary degrees of dulness by the term "flatness." It is the dulness of a brick wall rather than that of a table. In ascertaining the limits of dulness in cases of effusion, very light percussion should be employed, or the resonance of neighbouring parts will be elicited.

In cases of moderate effusion, in which the lung is texturally healthy, the upper margin of dulness in front is not a level line, but slants downwards and inwards in such a manner as to leave a somewhat triangular space of resonance (B, Fig. 19), the apex of the triangle being at the sterno-clavicular articulation. The resonance within this area is of

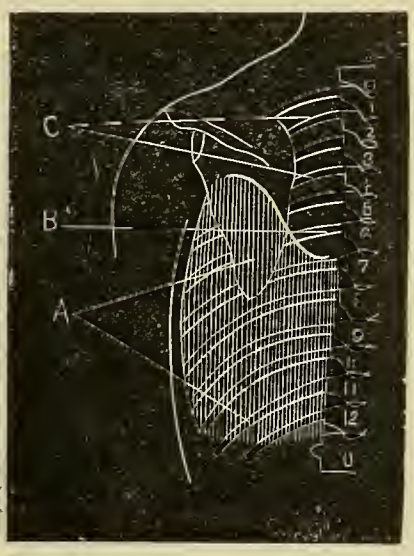

FIG. 20.—PERCUSSION SIGNS IN CASE OF MODERATE EFFUSION.

A, area of complete dulness ("flatness"); B, pulmonary note usually impaired; C, more or less tympanitic high-pitched resonance.

a peculiar tympanitic quality, termed "skodaic resonance," to which we shall again refer. The line of dulness (flatness) is highest in the axilla, and extends round to the scapula until in the interscapular region it again slants downwards, a tongue of comparative resonance protruding downwards in the postero-median line (B, Fig. 20). This curved line, which marks the upper limit of dulness, is sometimes spoken of as "Damoiseau's curve," or the "letter S curve of Ellis,"[13] and is in sharp contrast to the horizontal upper limit in cases in which air as well as fluid is present in the pleural cavity. The lower limit of dulness is again peculiar, the arch of the diaphragm being preserved (see Fig. 19), even when the

amount of fluid is large, so long, indeed, as skodaic resonance is manifested. This can be best observed in left-sided cases, and to it we shall again allude.

Even in cases in which the effusion is very considerable and of old standing, a triangular area of comparative resonance (but now somewhat tubular in quality) can still be made out at the sterno-clavicular angle, and also in the upper interscapular region. In cases of extreme effusion, however, the "flatness" of the percussion note extends throughout the utmost limits of the thorax, and these triangles of resonance are no longer to be found. In other cases, again, œdema or consolidation of the lung may obscure the signs over the areas referred to. As the fluid increases, the dulness encroaches upon the median line, extending farthest across at the level of the third and fourth cartilages, where it merges into the dulness of the displaced heart (D, Fig. 19).

2. *Displacement of heart towards the sound side* is the second cardinal sign of pleuritic effusion. The absence of this sign would negative the diagnosis of unilateral effusion, unless it were explained by special circumstances, such as fixation of the pericardium by old adhesions or consolidation of the opposite lung, the result of some former disease. But such cases are rarely met with, and the mediastinal folds are ready to shift to one side or the other, as the pleura becomes occupied and the lung on that side collapsed, leaving unopposed the traction of the other lung upon the mediastinum. Displacement of the heart as a sign of pleuritic effusion is of much greater value than displacement of other organs, because it takes place immediately and *pari passu* with the effusion. In this respect it strikingly differs from displacement of the diaphragm and abdominal viscera. The diaphragm is held up in the arched position until the effusion has mounted to a considerable height and the heart has already markedly retreated towards the opposite side. Consequently, displacement of abdominal viscera is no essential sign of pleuritic effusion, and is only present in extreme cases; displacement of heart, on the other hand, *is* an essential sign, and (unless prevented by countervailing causes) is present from the first in all cases of unilateral effusion.

Although the exact position of the heart is often somewhat difficult to make out, the trouble taken is well rewarded by

the importance of the sign. The position of the apex-beat should be carefully felt for by the hand, and, if necessary, by the ear through the medium of the stethoscope. Percussion may also be usefully employed to trace from the sound side the line of cardiac dulness.* The axis of the heart is not greatly changed in direction by any common degree of effusion. It becomes a little more vertical, and in very extreme cases it may even be slightly twisted; but we have never seen, nor been able to produce by experiment,[14] anything approaching to the complete turnover of the heart, which has been stated to occur in extreme effusion. In cases in which the heart is greatly displaced a systolic murmur is developed over its base, which disappears on removal of a portion of the fluid, and is presumably due to straightening of or slight traction upon the great vessels from pressure of the fluid.

3. *Absence of vocal fremitus* is a third very important sign of pleuritic effusion. It is obviously due to the intervention of a bad conductor of such vibrations between the vibrating media and the chest walls, and, if these two be united at any point by a cord of adhesion, the fremitus may there be felt. Just as light percussion is needed to define the exact limits of dulness, so light palpation is necessary, employing the finger-tips rather than the whole hand, in order better to exclude vibrations from above as the confines of the effusion are approached. With the same object the edge of one hand may be placed along the upper limit of dulness, whilst the other hand is employed in palpation below.

It will thus be observed that the three most essential signs of pleuritic effusion are elicited by palpation and percussion.

4. The stethoscopic signs in pleuritic effusions are of less importance, and are sometimes misleading. A certain value is justly attached to the *diminished breath-sounds*, and, except towards the upper confines of the effusion, to the *diminished voice-sounds*, in the diagnosis of pleuritic effusion, but in not a few cases of this disease the breath-sounds are audible over

* There is one fallacy with reference to cardiac displacement in the earlier stages of effusion that may be here noted—viz., that as the base of the lung retracts, the right or left margin of the heart (as the case may be right- or left-sided) becomes uncovered: this may lead to an apparent delay in the displacement of the organ, the extreme right or left margin thus being within reach of palpation.

the whole side, well defined, bronchial, and not easily distinguished from the breath-sounds of pneumonia. This is especially the case in children. The voice-sounds may also be well conducted over the whole side, and the whisper exaggerated even to pectoriloquy. As an instance we may quote the case of a boy aged eleven years, who came under the care of one of us at the Middlesex Hospital with acute pleurisy and effusion on the right side, of four days' duration. On the fifth day after admission the dulness had extended to the clavicle and well over the median line. The vocal fremitus was annulled, the heart was beating considerably outside the left nipple line, and the cyrtometer tracing showed enlargement of the affected side; but *bronchial breath-sound and whispering pectoriloquy* could be well heard posteriorly to the base, and somewhat more feebly over the lower two-thirds anteriorly. It was difficult to believe that one was listening over an immovably compressed lung, separated from the ear by some inches of fluid. Twenty-six ounces of clear serum were, however, readily removed the next day, with great relief to the little patient.

The reason why we should thus have very weak or absent breath-sounds in some cases and in others bronchial breathing demands a few moments' consideration. The weakening of the vesicular murmur is usually attributed to the interposition of a badly conducting layer of fluid between the lung and chest wall, and in slight cases this may be sufficient to explain the facts. But it must be remembered that fluid is by no means devoid of conducting power, as was shown, for example, in a case of left-sided pleural effusion under our care at the Brompton Hospital, in which an endocardial mitral murmur was well heard through the fluid below the angle of the left scapula, as well conducted as through lung. The presence of fluid alone, therefore, cannot be held to explain total absence of respiratory murmur, and it is probable that—in some cases, at least—this is attributable to attendant bronchitis leading to partial or complete occlusion of the bronchi by accumulation of mucus. The bronchial breath-sound when heard would seem to be due to the complete collapse of that portion of the lung auscultated, and the total elimination of its vesicular element. As a result, the laryngeal breath-sounds, provided the bronchi are patent,

are conducted unaltered through the fluid to the chest wall, some of their intensity being indeed lost during the process, but their special character remaining unimpaired.

Supplementary Signs of Effusion.—Much importance is usually attached to increase in the semi-circumference of the chest as a sign of pleural effusion. This is more apparent than real, and is chiefly noticeable during expiration, from the comparative absence of recession on the affected side. Should the effusion be considerable, the actual semi-circumference of the chest on the affected side will be augmented, and in any case in which a decidedly increased measurement is obtained we may be sure that the exudation is a large one. The *total* circumference of the chest must in any case of effusion be increased from the elastic outspring of the ribs.

Cyrtometer tracings give us more information than tape measurements, since they also take into account an altered shape, but, except for recording purposes, inspection with the unaided eye is more useful than either.

Intercostal fulness or *fluctuation* is a sign of exceptional occurrence in cases of effusion, intrathoracic pressure alone, except in very extreme cases, being insufficient to cause it. It is more commonly present in children than adults, and especially in weakly children with wasted muscles. A certain degree of laxity or inflammatory softening of the thoracic parietes is needed for the presence of this sign, which in our experience is more often associated with purulent than with serous effusion.[15]

Ægophony is a sign commonly met with in pleuritic effusions, but, as we have pointed out, may be sometimes also heard over consolidated lung (see p. 61). It will often be observed on listening for ægophony in cases of acute effusion that a certain lisp attends the voice-sound, which lisp is more sharply conducted than the ægophonic sound. In such cases, if the whispered voice be listened to, it will be found to be very distinctly conducted through the fluid (Baccelli's sign, see p. 120).

Pressure Signs.—It has been already pointed out that, whereas cardiac displacement is an essential sign of effusion, it is no evidence of intrathoracic pressure. Displacement downwards of the diaphragm is, on the other hand, always a sign of intrapleural pressure. The relationship of the dia-

phragm to the cavities above and below it is quite a different one to that which the mediastinum bears to the pleura on either side. We have on the thoracic side of the diaphragm, under normal conditions, a negative pressure; on the abdominal side the prevailing pressure is positive. The negative pressure within the pleura is maintained until the lung has completely retracted before the advancing fluid; after this point any further accumulation compresses the lung, and by its weight and pressure forces down the diaphragm. Hence displacement downwards of the abdominal viscera is a late phenomenon in pleuritic effusion. It also varies according as the patient has been keeping about, or has remained in bed, and it is more often met with in purulent than in serous cases.

The clinical outcome of these observations in regard to the displacement of the diaphragm is of great practical importance. As was first pointed out by Traube, and more fully explained in an admirable monograph by Dr. Garland,[16] of Boston, stomach note may be obtained at the sixth rib in the nipple line in the presence of a large effusion on that side. Similarly, on the opposite side, under the same conditions, the liver dulness may not be appreciably lowered. These facts are to be borne in mind in choosing the spot for paracentesis, otherwise awkward accidents may occur.

The period of effusion at which the intrathoracic pressure is converted from a negative or zero to a positive pressure upon the lung and heart is marked clinically (1) by the dulness mounting up above the third cartilage (the patient being in a sitting posture); and (2) by the skodaic resonance becoming changed from the full and clear tympanitic note to one of a more dull tubular quality, and finally becoming extinguished.

Skodaic resonance is thus of some clinical value in guiding us to a judgment as to the presence or absence of pressure within the thorax, and the advisability or urgency of interference by paracentesis. Different views have been held respecting the mechanism of its production in pleuritic effusion. But whether we adopt the view of Skoda himself and regard it as the relaxed lung note, due to the recession of the lung towards its root as the fluid advances, and resembling that of a healthy lung when percussed on the post-mortem table; or whether we accept one of the other varied explana-

tions given, it would seem that a slightly negative, or at least a zero, pressure is essential for the production of the full-toned Skoda note. We often meet with cases in a later stage of effusion in which, at or near the sterno-clavicular angle, the skodaic resonance is succeeded by a tubular or amphoric quality of resonance, resembling the percussion note over a considerable lung cavity, or over the cheek held on the stretch with the mouth slightly open. This resonance is probably produced by the lung being collapsed upon its root, and thus yielding the tracheo-bronchial resonance. Clinically it signifies a higher degree of effusion than the true skodaic note; it means that the lung is not simply contracted, but *collapsed* by the *pressure of the advancing fluid.* In the next degree of effusion all resonance of whatever kind is everywhere lost.

To the displacement murmur sometimes heard over the base of the heart in cases of intrapleural pressure we have already alluded. Another sign of more importance is the occurrence of crepitant sounds over variable portions of the sound lung. These congestion râles afford evidence of great stress of circulation in the other lung, and are associated with a congestive cough, and a viscid, sometimes blood-streaked sputum. With the development of such signs the dyspnœa becomes increasingly urgent, the surface dusky, and the patient is in imminent danger from syncope.

It is not usual, however, for the effusion to attain such dangerous proportions. In a large number of cases the area of skodaic resonance is not encroached upon, before the tide turns and absorption of the fluid commences.

In certain cases of pleural effusion, as pointed out by Grocco,[17] an area of dulness to percussion—the so-called *paravertebral triangle of dulness*—may be noted at the posterior base of the healthy lung. The apex of the triangle extends from the upper level of the effusion, whilst the sides are formed by the vertebral spines and by a line drawn from the apex of the triangle to the base of the thorax, some one to three inches from the middle line. The sign is not always present, and a similar area of dulness has been observed in certain cases of pneumonia. Its value appears to us to have been overestimated.

The fever of acute pleurisy commonly subsides in from one to three weeks, according to the severity of the attack (see

Charts, pp. 94 and 95). The tongue cleans, the appetite returns, and the patient has only to gain strength and to await recovery from the results of the local inflammation and effusion. In favourable cases as the fluid becomes absorbed, the lung expands from above downwards, the pleural friction sound returns, and with it usually some pleuritic pain, although of not nearly so intense a character as in the first instance.

Dr. Gee, in his work on Auscultation, minutely and accurately described the physical signs and other phenomena attendant upon receding effusions; suffice it here to say that the respiratory murmur slowly returns, at first in the upper portion of the chest, and the heart gradually resumes its normal position. The absorption of the last portion of fluid is often considerably delayed, and dulness and weak respiration may long persist at the posterior bases, with impaired movement and more or less contraction of chest. In some cases, indeed, these signs may never disappear, the pleura at the base having become thickened and adherent. In rare cases the fluid may show no signs of becoming absorbed, and the case may eventually become one of chronic pleural effusion (see p. 114).

Diagnosis.—The diagnosis of pleurisy is not attended with any great difficulty. As regards *pain*, it may be simulated by pleurodynia or intercostal myalgia, neither of which affections is, however, attended with febrile phenomena, nor with friction sounds, nor the signs of effusion. Pneumonia is the disease with which acute sero-fibrinous pleurisy is most often confounded; and we must observe that the two may coexist. The absence of rapidly developing bronchial respiration, with bronchophony and fine crepitation, the freedom from blood-stained sputa, and the gradual effacement of respiratory sounds and of vocal fremitus, together with increasing displacement of heart as dulness on percussion increases in extent, render the diagnosis of pleurisy with effusion certain.

Other conditions which may give signs somewhat resembling those of fluid in the pleura are collapse of the lung, especially in children, basal phthisis, bronchiectasis, new growth and hydatid of the lung. The diagnosis is in each case considered under its respective heading, but we may add that in most cases displacement of the heart to the healthy side—the cardinal sign of pleural effusion—is wanting. Only

in rare instances and under special conditions is the heart not displaced in the presence of acute pleuritic effusion.

The chief difficulty in diagnosis is to ascertain the probable *nature* of the pleurisy, whether sero-fibrinous or purulent. This subject we shall consider later (see p. 119).

Prognosis.—The immediate prognosis in the great majority of instances is decidedly good in cases of acute sero-fibrinous pleurisy, although the outlook in regard to completeness of recovery or otherwise is unquestionably influenced by judicious treatment. Sometimes the initial symptoms of pleurisy are very brief and slight, becoming rapidly obscured by effusion, and the true nature of the case is not recognised; perhaps advice is not even sought until the breathing be notably embarrassed by a large effusion. In these cases recovery is often slow and imperfect.

The danger of sudden death in cases of acute pleurisy with effusion is but small. It was exaggerated by Trousseau,[18] but is by no means to be left out of consideration, especially in cases in which the effusion reaches above the second rib—*i.e.*, in which there is positive pressure upon heart and lung. Whenever fine moist râles can be heard over the sound side, the patient is in some danger of heart failure with asphyxial œdema of the only working lung. Nor is the risk entirely to be ignored even in small effusions, for cases of this nature have been met with in which fatal syncope has occurred.[19]

We have seen some instances in which great agitation and dyspnœa, with threatened heart failure, have occurred in the early stages of rapid effusion, before any considerable amount of fluid has collected. Relief, however, has in these cases followed appropriate treatment by opium.

But the prognosis in a case of sero-fibrinous effusion deals with more than the immediate issue, since it includes the question as to the patient subsequently developing pulmonary tuberculosis. From what we have already said concerning the frequent association of tubercle with pleuritic effusion, this danger is a real one, which must not be forgotten.

Treatment.—The treatment of acute sero-fibrinous pleurisy of ordinary severity does not call for any very energetic measures. In the first stage of the disease our object is to reduce arterial pressure within the vessels of the diseased pleura—vessels which are temporarily paretic and in a state

of acute turgescence. One or two doses of aperient, together with saline diaphoretics and diuretics, are useful general measures to this end. At the first onset of the disease small doses of aconite may sometimes be given with advantage to reduce arterial pressure, but with the commencement of effusion this remedy should be stopped. Rest in bed must be absolutely enjoined, even in the least severe cases, and the diet restricted to nutritious fluids. There is no object to be gained by restraining the patient from taking bland drinks to ease thirst.

To lessen the intensity of the local lesion as judged of by pain and fever, the application of leeches to the side is often of great value. Hot poultices frequently changed are also beneficial, or the use of antiphlogistine, serving, by the dilatation of superficial capillaries, to ease pressure within the deeper branches of the intercostal vessels. A blister may sometimes be applied under the poultice, especially in those cases in which there is great pain. A fair amount of sleep must be secured by the judicious administration of opiates, which may be given in the form of Dover's powder, or in adults, if the pain be very severe, by subcutaneous injections of morphia. When there is great and alarming cardiac agitation in the early stages of the effusion, opium is distinctly indicated, and may be usefully prescribed with camphor, musk, or other diffusible stimulant.

The temperature of the patient and the signs of effusion must be carefully watched, although the former of itself never requires treatment.

Stage of Effusion.—Whilst the inflammatory fever is at its height the less we meddle with any effusion present—unless it become of itself a danger—the better. We must bear in mind that a certain amount of effusion is as much to be looked for in acute pleurisy as exudation into the air-vesicles in pneumonia, or "running at the nose" in nasal catarrh; and the products in the three cases do not, *cæteris paribus*, essentially differ. The pulmonary exudation consolidates *in situ;* the nasal product stiffens the handkerchief; and the exudation into the closed pleural sac, though remaining fluid, deposits a certain variable amount of coagulum, a thin layer of which covers and protects the roughened surface of the pleura, flakes of it also sometimes floating in the fluid. In other

cases irregular partitions of lymph form which span the pleural cavity, uniting the two sides and dividing the fluid into separate compartments (see Plate III.). Again, given acute inflammation of the coverings of the lungs, a certain amount of effusion is useful in separating and bathing in a bland fluid the inflamed and tender surfaces, and further, in keeping at rest the affected portion of lung. It is too often forgotten that the lung is in health exercising a constant traction upon the pleural sac, the vessels of which have therefore to sustain a negative pressure; this being so, it is but natural and physiological that if these vessels become temporarily weakened and congested by the inflammatory process, increased exudation should proceed from them. The effect of this exudation is to neutralise lung traction, and therefore to lessen afflux of blood to the weakened vessels. It is, moreover, the surest and most natural way of giving that rest to the inflamed surfaces which their recovery requires.

Fluid effusion being thus both natural and salutary in acute pleurisy, we must be watchful, but not meddlesome, in our treatment of its earlier stages. Up to the end of a week or ten days we need not, in ordinary cases, consider how to promote its removal; and, in the majority of cases, after this period the fluid will gradually subside by spontaneous absorption. At this stage counter-irritation is of undoubted value, and small doses of iodide of potassium may be added to the prescription. As the absorption is being effected, pain sometimes arises from the pleural surfaces again coming together. If severe, the temporary application of a broad band of adhesive plaster round the affected side will give relief. Mineral acid tonics, usually with iron, are now required, and, after complete subsidence of the temperature, change of air will hasten the reabsorption of fluid and the restoration of the function of the lung. For some years to come, however, the patient must remember that if he would avoid the danger of developing pulmonary tuberculosis he must be content to take matters easily, to enjoy long holidays, and to maintain his vitality at the highest pitch by means of a generous dietary and open-air methods of life. To spend the first winter at a high Alpine station is often a wise proceeding. If he be content to follow out treatment on these lines the outlook is good.

PLATE III

Paracentesis Thoracis.—Towards the end of the second or third week of illness, if absorption be not in fair progress, the question arises whether it be advisable to interfere and remove a portion of the fluid by paracentesis. Thanks to the labours of Trousseau, Hamilton Roe[20] (formerly Physician to the Brompton Hospital), Bowditch, Dieulafoy, and others, this operation has been rendered safe and easy of performance. The following propositions in regard to the operation appear to us justified by experience:

1. With good skodaic resonance down to the third rib, and with no material enlargement of the side, we may assume, as we have already pointed out, that, although much fluid be present, the lung is only held in the position of physiological rest. At an early stage of the illness, therefore, operative interference is not necessarily called for; and if pyrexia be still present under these circumstances at the end of the second week, it will be well to wait a little longer before performing aspiration.

2. If no progress in absorption has been made by the end of the third week, a portion of the fluid should, as a rule, be removed.

3. In cases in which the effusion mounts up to the second rib or higher, extinguishing skodaic resonance, and causing decidedly increased measurement of the side, we may be sure that there is positive intrathoracic pressure, impeding the heart's action, and compressing the lung so as to retard the circulation through it. In such a case the pressure may amount to 1 or $1\frac{1}{2}$ inches of mercury. Under these circumstances the patient is in danger of syncope. The signs especially indicative of danger—viz., lividity, palpitation, a straining retching cough, with frothy viscid sputa, sometimes streaked with a speck or two of blood, and the presence on the healthy side of rather fine crepitant râles—have been already pointed out. On the appearance of such signs, no matter what the stage of the disease, immediate interference is called for. The removal of two or three pints of fluid by the aspirator or syphon will give the patient immediate relief, and no further operation may be necessary. It was well pointed out by the late Lord Ilkeston[21] that the special symptoms of danger may sometimes be in abeyance whilst the patient is perfectly quiet.

4. In cases in which the effusion has occurred on the side on which there is already existing lung disease of an *advanced* tuberculous nature, we should be loath to interfere, as experience certainly shows that an effusion checks, and tends to arrest, the farther spread of the tuberculous process.

5. In cases in which there is any reason to suspect that the fluid may be purulent, quite apart from any question as to its amount, exploratory puncture must be at once resorted to.

Choice of Spot for Puncture.—Supposing paracentesis to be necessary, the first step is to select the best position for puncture. The physician is responsible in choosing the site for puncture, and must not share the responsibility with others. In choosing the spot he has to be sure that it is out of reach of diaphragm and heart, and that no lung is there adherent. The *sixth space in the mid-axillary line*, as a rule, best satisfies these conditions, and should be selected if, when tested by percussion, palpation, and auscultation, it prove satisfactory. This spot is most convenient for the following reasons: (1) it is most accessible whilst the patient is reclining in an easy posture; (2) the parietes are here moderately thin, and the intercostal space sufficiently roomy; (3) the mamma in the female is out of the way; and (4) it is sufficiently high up to be free from danger of perforating the diaphragm. (5) This point has also the advantage over that very commonly chosen, below the angle of the scapula, that the cannula is less likely to become blocked by flocculi, which tend, from the position of the patient, to gravitate towards the back of the chest. Most of the dry tappings which we have observed have occurred with the posterior puncture. (6) Lastly, this point is to be preferred to one chosen more anteriorly, as being more central with regard to the effusion.

In special cases of limited effusion the point for puncture must be decided upon accordingly, but it must be remembered that a central rather than the lowest point of the effusion should be selected.

Method of Performing Paracentesis.—The fluid may be removed from the chest either by simple syphonage or by means of an aspirator. If the *syphon* be employed, a simple trocar and cannula should be used, the lateral limb of the cannula being connected with a suitable receiver by means of a piece of elastic tubing two or three feet long, filled with

water previously boiled. In this way air is prevented from entering the chest during the respiratory movements. The elastic tube should be intercepted near the cannula by a piece of glass tubing, so that the nature of the fluid may be observed.

Before performing paracentesis, the greatest care must be taken to render both instruments and skin aseptic; otherwise organisms may be introduced and acute septic suppuration of the pleura be set up. The skin around the site for puncture should be carefully washed, and when dry painted with tincture of iodine or sponged over with some antiseptic solution. The trocar and cannula must be boiled.

Everything being prepared, and the patient reclining near the edge of the bed, with head and shoulders slightly raised, and leaning towards the affected side, a small area of skin at the spot chosen for paracentesis may be frozen with the ethyl chloride spray, to deaden sensibility. The first finger of the left hand should then be pressed into the intercostal space and the trocar inserted immediately over the upper border of the rib below, so as to avoid the intercostal vessels and nerves which lie under cover of the rib immediately above. By a sharp thrust it is then made to enter the pleural cavity. We may add that we have frequently performed the operation without employing any anodyne. In no case is a general anæsthetic necessary.

When the fluid has been struck, it may be allowed to run quietly until the flow ceases. The withdrawal by syphon is always gradual, and it is rarely necessary to stop the operation on account of cough or other trouble. The method is useful in cases in which the more convenient aspirator is not at hand.

If *aspiration* be employed, the form of instrument most convenient is that invented by Potain (Fig. 21), and it answers admirably. cork (*c*, Fig. 22), made of india-rubber, and of such dimensions as to fit accurately any ordinary wine or mineral water bottle, is perforated by two tubes, one of which *a'* is connected with the exit limb of the cannula. The second tube *b'* is adapted to an exhausting syringe. Each of these tubes is supplied with a stopcock, and the tube *a'* in connection with the cannula should extend somewhat farther into the bottle than *b'*.

Having fitted the apparatus, aspiration is sometimes performed as follows: the air in the bottle is exhausted, and then, the cannula tubing having been connected, the stopcock *a* is opened, when the bottle rapidly fills with fluid. The stopcock *a* is then again closed, the bottle detached and emptied, and the process repeated. By adopting this method of practice, however, we are employing quite an unknown suction power, which may amount to anything short of 15 pounds to the square inch, and which is not only quite unnecessary, but, if incautiously used, extremely dangerous, and especially so towards the latter part of the operation, when there is already negative pressure within the pleura.*

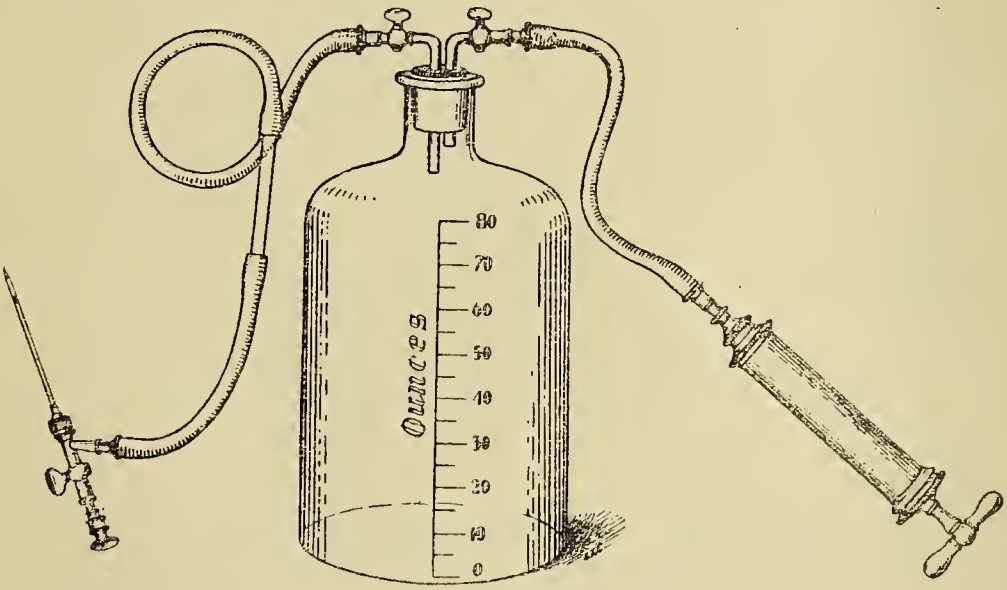

Fig. 21.—Potain's Aspirator.

A much more safe and useful method of employing aspiration is, not to exhaust the bottle, but to open the stopcock *a*, and to keep the syringe in just sufficient action to permit or encourage the flow of fluid through the exit tube *a'* into the bottle. When the bottle is filled, *the stopcock a must be closed* before the cork is withdrawn, otherwise there will be a rush of air into the chest.

Both the methods which we have described are very con-

* It would be very easy, of course, by a pressure gauge fitted into the bottle, to know at any moment what degree of aspiration is being used, but this arrangement would require a special bottle, whereas a great advantage of Potain's cork adjustment is that it renders any ordinary bottle available.

venient and safe for removing fluid from the chest, the syphon excelling the aspirator in the uniformity and measurability of the power employed, and also in simplicity and freedom from extra fittings. If a soft plug of fibrin block the cannula, it may be impelled through the tube by a momentarily increased aspiration power, or it may be dislodged by cautiously thrusting back the trocar.

The cannula used should have near the end a lateral as well as a terminal opening, the chance of its becoming blocked by a plug of lymph being thus lessened.

After a certain portion of fluid has been removed, varying from one to three or four pints, the patient begins to com-

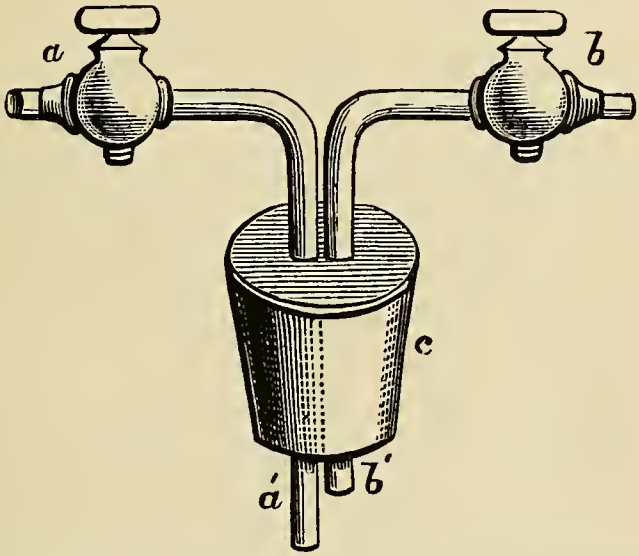

FIG. 22.—SHOWING CERTAIN DETAILS OF A POTAIN'S ASPIRATOR.

plain of oppression in the chest, and often experiences a pain below the cartilages, probably due to cramp in the diaphragm. This discomfort can be relieved by moderately firm pressure with the hand below the costal margin, but, together with a troublesome spasmodic cough which comes on about the same time, it indicates the presence of negative pressure within the thorax, and the necessity of caution in continuing the operation, which, if the symptoms persist, should be stopped. Indeed, it is quite unnecessary, even were it wise, to contemplate the removal of all the fluid, the withdrawal of a small portion leading often to a rapid absorption of the remainder, perhaps through lessening of pressure and opening up of the lymphatic vessels. If the warning be not

attended to, the patient is liable to develope that condition recognised as *albuminous* or *serous expectoration*,[22] in which, with straining cough, he brings up large quantities of strongly aërated sputum, resembling water whipped up with white of egg. The fluid contains albumin, and is, in fact, the product of acute œdema of the lungs. The condition is one of considerable danger, and will be dealt with more fully in a later chapter (see p. 354).

Chronic Sero-Fibrinous Effusion.

In the majority of cases of acute sero-fibrinous pleurisy recovery is effected by medicinal and local treatment, perhaps after paracentesis has been performed on one or two occasions. But patients are sometimes met with in whom, however often the fluid is removed, it invariably returns, and in whom the disease may therefore be termed "chronic." In certain other cases the initial symptoms are extremely slight, and the disease may be described as chronic from the first. In both classes the fluid remains serous, although occasionally, as in two cases which we have seen, a heavy deposit of *cholesterin crystals* may occur, sufficient to give the fluid a silvery or golden-spangled appearance, the colour varying according to the greater or less amount of blood present.[23] These chronic cases have become distinctly rare now that effusions are more efficiently treated in their earlier stages. Such patients, even with very large effusions into the pleura, may live for long with surprisingly little discomfort. Thus, some years ago, we had under occasional observation a policeman, stalwart-looking enough to frighten thieves, but who for four or five years had his left pleura full of fluid. In this instance, after a few partial tappings, it became evident that no expansion of the lung could be looked for, and, in the absence of any urgent symptoms, it was not thought prudent to submit him to any radical measure of treatment involving long illness, and probably a permanent thoracic fistula.

More recently, attempts, sometimes successful, have been made to deal with cases of this kind by aspiration, accompanied by the introduction of measured quantities of oxygen by means of the apparatus employed in performing artificial pneumothorax. In this way, as Mr. Morriston Davies[24] has shown, a larger quantity of fluid can be withdrawn, and a duly

registered negative pressure left within the pleura. As the oxygen becomes gradually absorbed a continual traction is made upon the collapsed lung, aiding its expansion, which in successful cases we have known to be complete. At the conclusion of the operation a drachm of sterile adrenalin chloride solution, 1 in 1,000, diluted in two or three times its bulk of normal saline solution, may also be injected into the pleural cavity with a view to diminish the secretion of fluid, as recommended by Sir James Barr.[25] It is needless to add that during these various manipulations the strictest antiseptic precautions must be observed.

Hæmorrhagic Pleurisy.

As we have already seen, in all sero-fibrinous pleurisies, the fluid exuded into the pleural cavity contains a limited number of red blood-corpuscles. These do not, as a rule, exceed 200 to 300 per cubic millimetre, and are insufficient, therefore, even to tinge the fluid. Sometimes, however, their number becomes so considerable that the liquid acquires a blood-stained and hæmorrhagic character, and may even contain 500,000 or more red corpuscles to the cubic millimetre. The colour of the liquid varies somewhat in different cases. It is bright red, if the blood has been recently effused; in cases of older date the tint may be red-brown, or even brown.

Trousseau[18] believed that malignant disease affecting the serous membrane was the common cause of such hæmorrhagic pleurisy, and the influence of his teaching has extended to the present day. Nevertheless, the statement is only partially true, for, apart from injury involving the fracture of a rib, it would be more correct to say that there are two causes, tubercle and cancer, either of which may be responsible for the condition. We must further remember that, although both cancer and tubercle are accountable for most of the hæmorrhagic pleurises which occur, yet neither of them is by any means essentially accompanied by an effusion of this nature.

The escape of the red corpuscles into the fluid may be due in some cases to the rupture of minute bloodvessels in recent adhesions, and in others to a toxic alteration in the vessel walls, whereby their permeability is increased. In the malig-

nant forms of the specific fevers and in certain blood diseases, such as scurvy, purpura and hæmophilia, the exudation may be thus blood-stained.

Clinically, hæmorrhagic pleural effusion does not present any special symptoms whereby it may be distinguished from other varieties of the disease, and it is, as a matter of fact, only recognised by exploratory puncture. The prognosis in each case will vary with the cause, and is not made more grave by the blood-stained nature of the fluid.

Hæmothorax, or the extravasation of pure blood into the pleural cavity, will be discussed in a later chapter (see p. 157).

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CHAPTER VIII

SUPPURATIVE PLEURISY

SUPPURATIVE pleurisy, or, to use its more ancient name, Empyema, does not differ essentially in clinical outline from the sero-fibrinous variety considered in the preceding chapter. As in the latter, the pleura becomes injected and inflamed, and effusion takes place, but, owing to the depressed vitality of the subject, the character and virulence of the micro-organisms concerned, and the consequent severity of the process, pus, not serum, is effused. The pus varies much in consistency, being thin as a rule in streptococcic cases, thicker and more "laudable" in those produced by the pneumococcus. Lymph is commonly found attached to the pleural surfaces, over which it forms a thick covering, and flecks of the same are seen in more or less abundance floating in the fluid.

Ætiology.—In considering the ætiology of suppurative pleurisy we are struck by the relation which it bears to age, the disease being much more common in childhood, whereas the chief incidence of the sero-fibrinous variety occurs between the ages of twenty and forty. Both varieties are more common in males than in females.

Clinically, we find empyema associated frequently with certain morbid states. It is thus very apt to occur in connection with pneumonia or gangrene of the lung, and when pleurisy occurs in such diseases as pyæmia, scarlet fever, typhoid fever and the puerperal state, it is mostly of the purulent variety. Sometimes it is secondary to disease below the diaphragm, such as liver abscess or gastric ulcer. In many cases, however, suppurative pleurisy is not a sequel to any other condition, but constitutes in itself the whole disease. In these cases the sanitary surroundings of the patient should be carefully investigated, as in many instances it will be found that defective drainage is really at the root of the matter.

The micro-organisms which are chiefly responsible for suppuration in the pleura are the pneumococcus and the streptococcus. In an examination of 137 cases in the hospitals of Boston and New York, Dr. Lord[1] found the pneumococcus present in 54 (39·4 per cent.); the streptococcus in 28 (20·4 per cent.); and the staphylococcus in 5 (3·6 per cent.). In 22 (16 per cent.) a mixed microbial flora was obtained, and in 25 (18·2 per cent.) the cultures were sterile, indicating possibly the presence of the tubercle bacillus, or more probably a pneumococcic infection in which the organism had already perished.

The original statistics of Netter,[2] to whom we are indebted for so much of our early knowledge of the bacteriology of pleurisy, pointed, in Paris at least, to the more frequent presence of the streptococcus in empyemata occurring in the adult. But this is not the case in England, and at St. Bartholomew's Hospital, in 32 consecutive examinations, the pneumococcus was found in 75 per cent., and the streptococcus in only 20 per cent. of the cases. Among children, Dr. Clive Rivière informs us that in 63 examinations at the East London Hospital for Children, he found the pneumococcus in pure culture in 55 cases (87·3 per cent.), once also in conjunction with the streptococcus, and once with the staphylococcus aureus. During the recent epidemic of influenza, however, the bacteriology has been exceptional, and the streptococcus has proved in the adult to be the predominant organism.

Occasionally such organisms as the influenza, diphtheria and typhoid bacilli, the bacillus coli, and members of the streptothrix group, are to be met with in the pus.

Symptoms.—Those of suppurative pleurisy do not differ strikingly from those of serous effusion, and in many cases it is impossible to make a certain diagnosis from symptoms and signs alone. The symptoms are as a rule less acute, but more adynamic in character than in sero-fibrinous pleurisy. Rigors or slight shiverings are of more frequent occurrence, and the daily oscillations of temperature tend to be more marked, and to continue week after week. The pulse is from the first more frequent, and keeps up its rapidity throughout the disease. The tongue is more coated, the general condition of the patient more depressed and anxious, and emaciation proceeds more rapidly. But perhaps the most important

sign of the effusion being purulent (although not absolutely characteristic) is the occurrence of hectic sweats whenever the patient falls asleep. Diarrhœa is a frequent, though not an exclusive, concomitant of pus in the pleura. Occasionally, the symptoms of empyema, especially in old people, are but slightly marked, and sometimes they may be wholly merged in those of some associated disease of the lung, such as pneumonia or phthisis.

Physical Signs.—These must necessarily be for the most part the same, whether the case be one of simple or suppurative pleurisy; but we may consider whether there are any physical signs which are characteristic of pus in the pleura, and by means of which it is possible to distinguish a purulent from a serous effusion.

Judging from our own experience, we may say that evident pointing, with fluctuation, is the only certain sign of the effusion being purulent. Œdema of the chest wall is very nearly pathognomonic, but has occasionally been seen in cases of sero-fibrinous effusion. But these are late signs, which with modern methods of treatment are now rarely seen. The course of the temperature is of itself of little assistance. It may not be higher than in serous pleurisy, and in rare cases there may be no attendant fever.

It was maintained some few years ago by Professor Baccelli[3] of Rome that by the mode of transmission of the whispered voice-sounds—*pectoriloquie aphonique*—the diagnosis could be made with certainty. In order to appreciate this sign, the unaided ear must be applied to some convenient point of the affected side, such as the dull region posteriorly below the angle of the scapula, and the patient directed to whisper some rough words, such as "trente trois" or "one, two, three." These will be well conducted to the ear if the fluid be serous; not so if it be purulent. Dr. Baccelli's theory to account for the difference of conduction in the two cases is that in serous effusions the fluid, being thin and homogeneous, transmits vibrations with facility; but the more the fluid departs from the homogeneous nature of serum, and the greater the number of corpuscular elements which it contains, the less complete is the conduction of sound through it. Experience, however, shows that, although of some use as an additional sign, this sign does not possess the crucial value

attributed to it by its distinguished advocate. Pectoriloquie aphonique is heard in perfection in some cases of pure sero-fibrinous effusion, but we have also heard it very well marked in certain cases of purulent pleurisy, and perfectly in cases of fœtid sero-purulent effusion.[4]

It has been suggested again, that the X-rays may be used to effect the diagnosis, some observers holding that the shadow cast by pus is denser than that produced by serum, and that the former completely obscures the ribs, whilst the latter does not. Here again, however, the rule is not absolute. In several cases of serous effusion (in the adult), which we have examined, we have found the outline of the ribs obliterated, and other observers have had a similar experience. In both forms of effusion the upper limit of the shadow when viewed upon the screen is often somewhat curved, the outer or axillary edge attaining the highest level.

Of more value in a doubtful case is an examination of the patient's blood. In empyema a leucocytosis is generally present, and the number of white cells may reach a very high figure. In sero-fibrinous effusions in the adult, on the other hand, unless otherwise complicated the leucocytes are usually normal in number or only slightly increased: a count above 15,000 is indeed altogether exceptional, and if observed would point to empyema. In children, however, a leucocytosis is sometimes present even in sero-fibrinous cases.

From what we have said it will appear that the distinction between a purulent and a serous pleurisy must in many cases be a matter of considerable difficulty. The exploring syringe should therefore be used in all doubtful cases as a matter of routine. Too fine a needle must not be employed, or the pus, if thick and viscid, as in many cases of pneumococcic infection, may not be withdrawn. But even with a large needle it sometimes happens that a shred may block the orifice and the puncture be apparently without result. It is wise, therefore, in all such cases, after withdrawing the syringe, carefully to examine any material contained in the terminal end of the cannula. In this way we may sometimes prove that pus has really been struck, though at first sight nothing may have been withdrawn. Strict antiseptic precautions must in all cases be employed.

Pulsation of the Fluid.—There is one other physical sign which, though not invariably restricted to purulent cases, is

yet, when present, far more often met with in this variety of effusion, and may therefore be best considered here. We refer to pulsation communicated to the fluid by the heart's action, and in some cases closely simulating that of aneurism. The phenomenon was first described in 1844 by MacDonnell, of Dublin, a pupil of Graves and Stokes, and is of great rarity. To cases exhibiting it the name *empyema pulsans* has been applied. The sign is not restricted to any one variety of empyema, but is observed for the most part in very chronic left-sided cases, in which the effusion is large. The pulsation may manifest itself only in a portion of the empyema which has penetrated the chest walls; and in this case it is more definitely expansile in character. In other cases the impulse is not confined to any definite tumour, but may be felt over a limited area of the chest, most commonly in the antero-lateral region of the left side.

The cause of the phenomenon is not very clear. In some of the cases pericarditis with effusion has been present in addition to the empyema, and Traube explained the condition as being due in part to suppurative softening, with increased extensibility of the costal pleura, and in part to the presence of pericardial effusion favouring the transmission of the heart's impulse. But in the light of the following cases such explanations are insufficient.

Some years ago the late Professor Maclean was kind enough to allow one of us to examine at Netley a soldier who had a very excessive effusion into the left pleura, and who had so marked a pulsation in the left supramammary region as to cause some hesitation in performing paracentesis, lest there should be a large aneurism in addition to the hydrothorax. A right judgment was, however, arrived at, and a large quantity of *serous* fluid having been removed, the doubtful sign of aneurism disappeared. There was no evidence of pericarditis in this case.

Another case of effusion, right-sided and of older standing, came under our notice at the Brompton Hospital, in which a rhythmic impulse conveyed through the fluid suggested aneurism as a complication. The fluid was removed by several tappings, and the symptoms of aneurism disappeared. The liquid was somewhat opaque and at first slightly blood-stained.

From these cases it is clear that neither suppuration in the sac nor pericarditis is essential for the production of pulsation, and probably nothing more is needed to account for the phenomenon than an amount of fluid, of whatever kind, which

shall exercise a certain degree of pressure (neither too much nor too little) upon the heart.

Another very rare pressure effect which we have observed in two or three instances is an altered quality of voice and cough. A husky voice and a laryngeal quality of cough, undistinguishable from that so often heard in cases of mediastinal tumour or aneurism, gave rise in these cases to great doubt as to the diagnosis, yet both phenomena disappeared after paracentesis.

Special Varieties of Empyema.

1. **Fœtid Empyema.**—It sometimes happens that when fluid is drawn off by exploratory puncture from the chest it is found to be extremely fœtid in character. This is due to the presence of micro-organisms, which produce by their growth certain evil-smelling substances. In some cases bacillus coli has been found, in others various putrefactive organisms, some of them anaerobic in character. No one organism is therefore responsible for the condition.

This variety of empyema may be primary, and is then not uncommonly encysted, or it may follow upon some disease, such as pulmonary gangrene or bronchiectasis. We have seen it also rapidly bring to a fatal termination a case of chronic hydropneumothorax. The symptoms, though somewhat more severe, do not differ materially from those of ordinary empyemata. Indeed, the nature of the disease is generally revealed by the exploring syringe.

The prognosis must depend largely upon the cause, but, provided there be no serious lung disease in the background, and early operation be resorted to, the outlook of the case is not more grave than that of a simple case of empyema.

2. **Tuberculous Empyemata.**—Suppurative pleurisy sometimes attacks a patient already the subject of phthisis, and is then usually due to one or other of the pyogenic organisms which are commonly responsible for the disease. Occasionally it happens that the empyema results from the action of the tubercle bacillus alone, and such cases present certain peculiarities. Usually they are of an exceedingly chronic type, the presence of fluid within the chest being discovered in some cases almost by accident. In others the disease commences as a sero-fibrinous pleurisy, and after one

or two tappings the fluid withdrawn, at first clear, becomes gradually opalescent and then sero-purulent, even in the absence of other organisms than the tubercle bacillus. In either case the symptoms are very slight. There may be no fever, and the only complaint is of slight dyspnœa on exertion. The fluid, as we have indicated, is commonly rather sero-purulent or opalescent than truly purulent.

The patient may live for years, death finally ensuing from the supervention of tuberculous disease elsewhere, or through some intercurrent malady.

Very occasionally, as in a case recorded by the late Sir William Osler,[5] suppurative tuberculous pleurisy may run an acute course.

The patient was a woman, twenty-one years of age, whose illness set in abruptly with shivering and severe pain in the right side, and was accompanied by high and irregular fever until her death six weeks later. At the autopsy the right pleura was found thickened and infiltrated, and to contain between its adherent surfaces numerous small pockets of pus. Tubercle bacilli were extraordinarily abundant in the pus, and cultures proved the absence of other micro-organisms. In addition to the pleurisy, recent miliary tuberculosis of the lungs, spleen, and kidneys was found.

Such cases are exceedingly rare.

3. **Localised or Encysted Empyema.**—We have spoken hitherto of the signs and symptoms of suppurative pleurisy affecting the general pleural cavity, but we must remember that the disease may be limited in its extent, giving rise to special symptoms, according as it is *interlobar, mediastinal,* or *diaphragmatic.*

(1) In the *interlobar* variety the pus is contained within one of the fissures of the lung, the edges of the fissure being glued together as the result of the inflammation. The symptoms, such as cough, fever, dyspnœa, will suggest disease of the respiratory organs, but the absence of definite physical signs will render the diagnosis uncertain, until, as not uncommonly occurs, rupture into a bronchus and the sudden expectoration of pus reveals the nature of the disease. In such cases the X-rays are of assistance, revealing the presence of a rounded shadow within the chest, which, from the circumstances of the case, is strongly suggestive of encysted fluid.

(2) The inflammation is sometimes limited to the pleura covering the mediastinal surface of the lung. Such *mediastinal*

pleurisy may affect the region of the anterior mediastinum, when the signs often suggest a large pericardial effusion. More commonly the posterior mediastinum is attacked, when symptoms of pressure upon the œsophagus and trachea, developing rapidly and brought to an abrupt close by the sudden expectoration of pus, are the chief signs.

(3) *Diaphragmatic Pleurisy.*—This variety we have already discussed in dealing with simple pleurisy (see p. 88).

Course and Termination of Untreated Empyemata.—A few cases are on record which show that occasionally an empyema may undergo spontaneous absorption and the patient completely recover. The examples have been mostly cases of pneumococcic pleurisy in children. Such a fortunate termination is, however, exceedingly rare, and must not be expected.

If left untreated, an empyema may prove fatal in a short time from septicæmia or other septic complications, such as pericarditis, or, more rarely, cerebral abscess. In other cases it will rupture through the lung, with perhaps immediately fatal results from flooding of the bronchial tubes, or discharge in an intermitting manner and, in a certain proportion of instances, terminate in recovery. On the other hand, the pus may burrow through the chest wall, and point externally. This usually occurs in more chronic cases, which are now happily but rarely seen, although, before early operation was resorted to, they were common. The empyema points perhaps most often in the fifth space anteriorly, in the nipple line, but may do so in other situations, both in the front and back of the chest. After a short time the skin gives way and the pus is evacuated, but the drainage is rarely satisfactory and suppuration continues. The patient as a rule succumbs to lardaceous disease, recovery in these untreated cases being rare.

Cases have occurred in which an empyema has burst into the pericardium, peritoneal cavity, or the œsophagus. In other instances the pus has entered the sheath of the psoas muscle and formed a psoas abscess, or, as Mr. Stephen Paget[6] and others have recorded, has pointed in the lumbar or gluteal region. In other cases again a perinephritic abscess has been simulated. To this variety of the disease the term *migrating* or *wandering empyema* has been applied.

Diagnosis.—In a case presenting the cardinal signs—dul-

ness, absence of vocal fremitus, and displaced heart—with other subsidiary signs indicative of fluid in the pleura, and presenting symptoms suggestive of the effusion being purulent, the use of the exploring syringe forms the crucial test. It is not a little surprising, however, in actual practice to find how difficult it is sometimes to strike the pus-containing cavity. In such cases it may be necessary to give an anæsthetic and to explore deeply at several points over the dull area. The rules of procedure are the same as for simple effusion (see p. 110).

If pus be obtained, there is still the possibility in exceptional cases of the abscess cavity being in the lung instead of in the pleura. Or, again, it may be a subphrenic or a liver abscess pushing up the diaphragm, and thus encroaching upon the thoracic space, and leading to collapse of the base of the lung. Complete dulness of note up to the angle of the scapula, or even higher, with weakened or bronchial breath-sound and diminished vocal vibrations, may be thus produced, whilst the heart is not infrequently displaced. If the exploring needle be inserted in the axillary region through the sixth or seventh space, pus will very probably be evacuated, the needle passing through the displaced diaphragm and striking the fluid below it. We can recall a case of this kind in which complete dulness extended up to one inch above the angle of the right scapula, accompanied by weak bronchial breathing and diminished vocal vibrations, and in which the apex-beat of the heart was in the fifth space, 1¼ inches outside the nipple line. Forty-seven ounces of pus were withdrawn, under the impression that the case was one of empyema. It was only when resection was performed later, and when the pleura was found to be free from fluid and the diaphragm bulging into the pleural cavity, that the true nature of the disease was revealed. In this case an X-ray examination would have been of value.

The observer may suspect the presence of subphrenic abscess (which may, however, be complicated by the presence of a pleural effusion) if, in addition to the physical signs of fluid in the pleura, abdominal symptoms, such as tenderness and distension over the region of the liver, or œdema of the skin in this situation, be present. But sometimes, as in the case above referred to, these are absent, and the diagnosis is

only effected when the position of the pus is accurately located by surgical intervention. As a rule, the previous history and the presence of abdominal symptoms will suggest a special scrutiny in such cases. For a consideration of the condition known as *pyo-pneumothorax subphrenicus*, when the abscess cavity beneath the diaphragm contains air as well as pus, we must refer the reader to the next chapter (see p. 149).

Prognosis.—From what we have said as to the ætiology of empyema, it will be evident that the prognosis of a case must depend to a great extent upon the cause. Thus it is clear that an empyema complicating a case of puerperal infection must have a different outlook to that of one occurring in a patient previously in good health. In the latter case the prognosis is nearly always favourable, provided that adequate surgical treatment be early resorted to; but the longer this is delayed the more grave does the outlook become, as regards both the immediate safety of the patient and his chance of complete recovery.

In cases of empyema following pneumonia or typhoid fever the prognosis is generally satisfactory, partly perhaps because such cases are as a rule detected and treated early.

In children the prognosis depends to some extent upon the age, the mortality increasing as the age of the child diminishes. In all cases the prognosis of a pneumococcic pleurisy is better than that of one of streptococcal origin.

Treatment.—The treatment of suppurative pleurisy does not differ essentially from that of other purulent collections. The pus must be evacuated as speedily and thoroughly as possible, and its reaccumulation prevented by the provision of free drainage. This should be effected by the incision of an intercostal space, or by the resection of a rib. If the effusion be very large, it is wise as a preliminary to incision to remove a portion of the fluid by aspiration, since the sudden evacuation of a large quantity of liquid may sometimes produce fatal syncope.

The operation for empyema should never be performed without first inserting the exploring syringe. For lack of this precaution we have known a rib resected in a case which proved to be a rapidly-growing sarcoma of the lung, which had worked its way to the surface and suggested a case of pointing empyema.

Should the collection be small, the incision must be made immediately over the seat of the localising puncture. But in the case of an empyema filling to a large extent the general pleural cavity, the site chosen should be over the ninth rib, just outside the angle of the scapula. Here, as Sir Rickman Godlee[7] has pointed out, owing to the adhesion of the diaphragm to the lower ribs the opening soon leads into the most dependent portion of the pleural cavity, and satisfactory drainage, both in the sitting and lying posture is secured.

Whether incision alone or incision coupled with resection of a portion of a rib should be performed is a matter on which there is some difference of opinion. In our own view resection is as a rule to be preferred, since when incision is alone resorted to, the drainage-tube frequently becomes nipped from the falling together of the ribs; but if in a given case the patient be very ill, and the avoidance of shock all-important, we should not hesitate to advise a simple incision. On the other hand, if the side be fallen in and the intercostal spaces much diminished, resection is absolutely necessary.

The anæsthetic given should be gas and oxygen or chloroform, not ether, the latter being rarely suitable in cases of chest disease, owing to the irritation of the respiratory passages which it so frequently produces.

In performing resection an incision some two to three inches in length should be made along the course of the rib selected down to and including the periosteum. This should then be separated from the rib by a suitable raspatory, and about two inches of the rib removed by bone forceps. Having stanched all bleeding, the knife is then inserted through the costal pleura, the artery being carefully avoided, and the incision freely enlarged by the introduction of the finger or by opening out the blades of a dressing forceps. The pus thereupon escapes. The extent of the abscess cavity should be examined by the finger, and any large masses of lymph which may be loose and within reach should be removed. If the chloroform be now sufficiently slackened, without allowing the patient to become quite conscious, the irritation of the pleura by the finger or by the admission of cool air will give rise to considerable coughing, which is most salutary in leading to the more effectual expulsion of such coagula before the introduction of the drainage-tube. After the operation

a thick dressing should be applied, and the patient returned to bed.

Washing out the pleural cavity, whether at the time of the operation or subsequently, is not as a rule to be recommended. It is rarely required, because after free drainage even fœtid empyemata rapidly cease to be offensive; it is not to be recommended, except in the rare event of the pus remaining fœtid, because the procedure is not devoid of danger, instances of sudden death from *pleural shock* having been recorded, though the recent experiences of war surgery would suggest that this danger has been exaggerated. Should the special circumstances of a given case require its use, care must be taken that the irrigation be performed very gently, the danger to the patient being in this way materially diminished.[8] In some cases, especially in children, immersion in a warm iodine bath (ʒi. of the strong tincture of iodine to the gallon of water, so that the liquid assumes a light sherry colour) may be employed as a safe and satisfactory method of irrigating the pleura.

The after-treatment of the case is of great importance, the same antiseptic precautions being necessary until complete closure of the wound is effected.

During convalescence, to assist expansion of the lung, various breathing exercises may be performed. The attempt to expand collapsed and adherent lung sometimes advocated, by blowing against resistance is contrary to Nature's method, and unless very gently employed tends to produce emphysema rather than healthy lung expansion. Mild calisthenics and breathing exercises carefully carried out, and mountain air are to be advised.

In some cases in which the lung is diseased, or the empyema has been too long neglected, the lung may fail to expand. The chest wall then falls in to some extent, but, being unable to meet the lung, a large pus-secreting cavity remains. In cases which are not tuberculous in nature, expansion of the lung may sometimes be effected by the operation devised by M. Delorme,[9] and known as *Decortication of the Lung.* This consists in the removal of sufficient area of rib, and the peeling off of the layers of false membrane which overlie the visceral pleura and bind down the lung, thus enabling it to expand, which it sometimes does with great rapidity.

Should decortication prove impossible, some form of *thoracoplasty* must be undertaken. This operation consists in removing those portions of the ribs which bound the cavity, thus allowing the chest wall to be brought in contact with the lung surface. Such a procedure is usually termed an *Estlander operation*,[10] though the distinguished surgeon of Helsingfors was not, as a matter of fact, the first to perform it. Should the parietal pleura bounding the cavity be found densely thickened and cartilaginous in hardness, the pleura and intercostal muscles should be removed as well (Schede's operation): the overlying soft parts then fall in, and constitute in future the non-rigid chest wall over the area dealt with. Such operations lead to much deformity, and naturally vary much in severity, according to the size of the cavity to be dealt with. In the more serious cases it may not be possible to do all that is required at a single operation. But if the lung shows no sign of expanding, if the cavity be large, the discharge considerable, and the patient's general strength permits, operative interference on the above lines is to be advised. From it improvement generally results and, not infrequently, recovery, in cases which would otherwise steadily go downhill.

Should only a small sinus remain, an attempt may be made to hasten its closure by placing the patient upon open-air lines of treatment or by a stay at some breezy East Coast resort, such as Margate, before proceeding to more drastic measures of treatment. Closure of the sinus may sometimes be accelerated by the employment of a few doses at weekly intervals of a vaccine prepared from the specific organism originating the disease.

In the rare cases of *double empyemata*[11] it is well at first, while incising one pleura, to perform aspiration only upon the other. In this way time may be gained, and the more radical treatment of the opposite empyema undertaken at a later date, if it becomes necessary. Nevertheless, we have seen the two radical operations successfully performed within a very few days of one another.

Such is the line of treatment which we should usually recommend in cases of empyema. In the *recent epidemic of influenza*, however, numerous cases of empyema were met with during the acute stage of the disease, and associated

with an underlying broncho-pneumonia. Most of these were, as we have indicated, streptococcal in nature, and the pus thin, watery, and blood-stained, and in some cases only to be recognised as pus by the formation of a deposit on standing, which proved microscopically to consist of polymorphonuclear leucocytes. The patients were for the most part exceedingly ill from influenza, and the mortality following resection and drainage was high. Aspiration, repeated when necessary, proved more successful in these exceptional cases. We had one case also under our care in which, after aspiration had been considered and negatived, the fluid was spontaneously absorbed, and the patient made a complete recovery.

The treatment of *empyema occurring in the course of pulmonary phthisis* requires careful thought in each individual case. The lung, being diseased, does not expand when the fluid is removed so readily as a healthy lung; and, again, there is reason to believe that the pressure exerted by the fluid upon the lung diminishes its blood-supply, and tends to check the further spread of the tuberculous disease. We shall not be so ready, therefore, to proceed to operative treatment without first considering all the circumstances. If the bacteriological examination prove the empyema to be pyogenic in nature, and if it be acute in type, incision or resection must be performed. If, on the other hand, the pulmonary disease is extensive, and the empyema cause but slight symptoms, then it will be wise to forbear, resorting to aspiration from time to time, as occasion demands.

In cases of *true tuberculous empyemata,* which, as we have seen, are mostly of chronic type and of fair prognosis, experience shows that operative treatment may lead to hectic, and but hasten the end. An occasional paracentesis, with or without oxygen replacement[12] (see p. 114), should therefore, as a rule, be our line of treatment in these cases, and it is gratifying to note how slowly the fluid sometimes returns.

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CHAPTER IX

PNEUMOTHORAX

Hydro- and Pyo-Pneumothorax.

THIS disease was practically unknown until Itard,[1] in the year 1803, drew attention to it in his graduation thesis at the University of Paris, and designated it "pneumothorax." The succussion splash, it is true, was known to the writers of antiquity, but Hippocrates and his disciples regarded it as a physical sign, not of air in the pleura, but of empyema.* In the Middle Ages, though certain writers refer to the occasional presence of air in the pleural cavity, the occurrence was regarded as a phenomenon rather than as a definite disease. The credit must, therefore, be given to Itard for his recognition of the malady, and for emphasising its connection with tuberculosis. It was Laennec, however, who first rendered its diagnosis easy, and his name must ever be associated with the disease.

Ætiology.—Laennec[2] believed that pneumothorax might be both non-perforative and perforative in nature. In the former case, termed "simple" or "essential" pneumothorax, he thought that the air was either secreted by a perfectly healthy pleura, or was the result of decomposition of fluid, generally purulent in nature, previously effused into the pleural cavity. But such cases he regarded as somewhat rare, and he believed that the commonest cause of the condition was the softening of a superficial tubercle or the rupture of a vomica, whereby a communication between a bronchus and the pleural cavity was established, thus leading directly to the entrance of air.

These views were for a time accepted, but in an able article

* "Not to recognise when practising succussion the presence of an empyema . . . shows a lack of skill" (Hippocrates, *De Morbis,* i., § 6).

in the *Gazette Hebdomadaire* for 1864 Jaccoud maintained that there was no sufficient proof of the occurrence of pneumothorax from the secretion of air through a healthy pleura, and expressed serious doubts as to the presence, save, perhaps, very occasionally, of air in the pleura derived from the decomposition of liquid effusions. Jaccoud contended that in cases of so-called "simple" or "essential" pneumothorax a minute perforation of the pleura probably occurred, the aperture of which had subsequently closed. There can be little doubt that this criticism is just, and that—apart from gunshot wounds of the chest, in which, as we shall see (p. 160), hæmopneumothorax is common, the air being often then produced by the growth of anaerobic gas-forming organisms introduced with the bullet or by fragments of clothing—pneumothorax is nearly always perforative in character.

The conditions under which a rupture of the pleura and the formation of pneumothorax may occur are very varied. The following table, giving the results obtained by Biach[3] from an analysis of 918 cases in the hospitals of Vienna, shows what the conditions are, and roughly their relative frequency:

Pulmonary tuberculosis	715
Gangrene of the lung	65
Empyema	45
Wounds	32
Bronchiectasis	10
Abscess of the lung	10
Emphysema	7
Breaking down of hæmorrhagic infarct	4
After paracentesis	3
Hydatid of the lung	1
Rupture of bronchial gland into bronchus and pleura	1
Caries of the ribs	1
Caries of the sternum	1
Abscess of the mamma	1
Perforation of the œsophagus	2
Perforation of a gastric ulcer into the pleura ...	2
Rupture of hydatid of liver into colon and pleura ...	1
Rupture into the pleura of a sacculated peritoneal effusion	1
Cause undetermined	16
Total number of cases	918

The above figures refer to civil life and demonstrate clearly that, excluding gangrene of the lung, wounds of the chest, and the discharge of empyemata, it is rare for pneumothorax to occur from any other cause than the breaking down of subpleural areas of tuberculous consolidation. It is doubt-

ful, indeed, whether the figure 77·8 per cent. estimated by Biach really does justice to the great predominance of phthisis. Possibly 90 per cent., as given by other writers, is a truer estimate. In the recent war shell and gunshot wounds of the chest naturally took the chief place in the ætiology.

In regard to empyema, it should be noted that in by no means every case in which perforation of the lung occurs does pneumothorax result. This is accounted for by the viscid pus which obstructs the bronchus, and perhaps by the often fistulous character of the opening which impedes the entry of air.

The cohesion of the pleural surfaces, like that of any other plane surfaces in airtight application, is very considerable[4]; hence, when both pleuræ are wounded, as by a fractured rib or perforation from without, the air will not infrequently escape from the lung into the subcutaneous cellular tissue of the thoracic wall rather than into the pleural cavity.

In whooping-cough rupture of the lung is occasionally produced by the violent expiratory efforts characteristic of the disease. But in these cases the pleura generally remains intact and the air is effused into the subpleural tissue of the lung, sometimes finding its way eventually into the cellular tissue of the neck and chest walls. Subcutaneous emphysema, not pneumothorax, thus as a rule ensues. To this class would probably belong the case of a young lady who, during an attack of asthma, was found by one of us to have emphysematous crackling throughout the cellular tissue of the neck, pectoral region and back.

Another cause of pneumothorax to which we may refer is emphysema. Cases referable to this disease are undoubtedly rare, but a few verified by post-mortem examination are on record. In the case recorded by Sir J. Kingston Fowler[5] the patient died suddenly shortly after labour, and at the autopsy no sign of tubercle was found, but only a ruptured emphysematous bulla. Similarly, in the case described by Dr. Ranking,[6] in which, three months after recovery from pneumothorax, death occurred from a ruptured dissecting aneurism of the aorta, no lesion in the lung except emphysema could be seen. These cases are, however, exceptional, and we must not assume when pneumothorax occurs suddenly, and in the absence of physical signs of disease of the lung, that a

ruptured emphysematous bulla has been the cause. In the great majority of such cases a minute tuberculous lesion has been present, and to its rupture we must trace the complication.

Tuberculous Pneumothorax.

The constant breaking down of caseous material in the course of phthisis tends, by undermining the pleura and interrupting its vascular supply at certain points, to cause it to soften and to give way as the result of some trifling increase of air-pressure during cough or effort. The perforation may occur at any period of the disease; even at the very commencement a small subpleural tubercle may soften and break through the pleura, but such an occurrence is much more common in the later stages.

In the majority of cases of pulmonary tuberculosis, pleurisy of a dry and adhesive kind accompanies and keeps pace with the progressing lung consolidation. But it may be observed post-mortem in some cases which are marked by acuteness of process, and by the pneumonic character of the consolidations, that no adhesion between the pleural surfaces has taken place, each surface—the visceral pleura more especially—being covered by a thin finely granular semi-transparent layer of lymph, which may readily be scraped off with a knife, leaving the shining, almost healthy-looking pleura denuded. At certain points of the surface thus affected will be seen opaque yellow spots, varying in size from that of a pin's head to that of a bean, some close to the pleura, others more dimly seen through it (Plate IV.). On making a vertical section through such an opacity, we divide a more or less softened caseous nodule, having its centre, perhaps, already excavated. It is at some one or more of these points that the pleura most commonly gives way.

Pneumothorax is thus most likely to occur in the more acute pneumonic forms of phthisis, and least so in the more chronic fibroid varieties of the disease. Nevertheless, it is sometimes met with in the latter, and it was pointed out by one of us[7] that sinuses, in no respect differing from those which originate from chronic abscesses or carious bone, may sometimes be found extending from old cavities towards the surface. Such sinuses may penetrate both layers of the

PLATE IV

PNEUMOTHORAX

The drawing shows the posterior portion of the lower lobe of the left lung. Over the upper lobe the pleural layers are adherent, and at A the parietal layer is shown cut across and having its edge turned back. Two inches below the apex of the lower lobe, at B, the visceral pleura is seen to be thin and necrotic, and in the centre a perforation has occurred, the aperture of which admitted a small probe. At C the pleura is also becoming necrotic.

From a girl aged sixteen, who died from acute caseous tuberculosis of four months' duration. The pneumothorax gave no sign during life, and was discovered at the autopsy. The pleural cavity contained air and three ounces of clear serous fluid.

(From the Brompton Hospital Museum. Natural size.)

PLATE IV

Pneumothorax, showing Small Point of Rupture in Pleura.

To face p. 136.

pleura, and open into the subcutaneous cellular tissue of the thoracic wall, or if, as is rarely the case, the pleura be not firmly adherent, a communication with the pleural cavity becomes established. In one remarkable case described in the paper referred to a sinus had opened from an old cavity through the posterior mediastinum into the opposite pleural space. When a communication is thus effected between a more or less deep-seated vomica and the pleural surface, the perforation is so oblique or sinuous as to be always practically valvular.

Concerning the frequency of pneumothorax occurring in the course of well-marked phthisis, we may state that of 1,069 post-mortem examinations on this disease made at the Brompton Hospital during the years 1887-1894, 88, or 8·2 per cent., showed this lesion.[8] Our own post-mortem figures at the same hospital during the three years 1900-1903 yielded a somewhat lower result, pneumothorax being observed in 17 out of 263 cases, giving a percentage of 6·4.[9a] Allowing for the extra zeal with which permission for an autopsy is naturally sought in an interesting case, we shall probably not greatly err if we assume that some 6 per cent. of all cases of phthisis die with pneumothorax.

The complication occurs somewhat more frequently on the left than on the right side, the figures given by various writers being as follows:

	Number of Cases.	Right Side.	Left Side.
Louis[10]	8	1	7
Walshe (collected)[10]	85	30	55
Samuel West[11]	83	41	42
The Authors[10]	39	16	23
,, ,, [9b]	19	7	12
	234	95	139

The greater vulnerability of the left lung which these figures disclose is probably only in conformity with the increased frequency with which this lung becomes the seat of tuberculous disease.

The opening into the pleura is generally single, but if softening is taking place rapidly two or more perforations

may be found. Of seventeen autopsies made by one of us, fourteen showed a single point of rupture, two a double perforation, and in one case there were three.[9b] More rarely the pleura over a limited area has been found necrotic and riddled with holes. As a rule no difficulty is experienced in finding the perforation. On opening the chest the lung is seen to be collapsed and lying along the vertebral column, though perhaps with its apex still retained by adhesions in contact with the chest wall. The surface of the organ is lustreless and somewhat more opaque than usual. In order to locate the point of rupture, the pleural cavity should be filled with water and air pumped into the trachea by means of a pair of bellows. The bubbling out of the air at once reveals the perforation.

The size of the opening varies from that of a pin's point to a threepenny-piece (see Plates IV. and V.), but usually it is small and circular. In the majority of our own observations the aperture admitted only a small probe, but in two cases it was the size of a pea and a threepenny-piece respectively. On the other hand, the aperture may be occasionally linear rather than circular, and in two instances which we have recorded it was found to measure respectively $\frac{1}{5}$ and $\frac{1}{3}$ inch in length.[9b]

The position of the opening is very variable. It is usually found in the upper half of the lung, though rarely higher than the third rib, since above this point the pleural cavity is generally obliterated by adhesions. We have ourselves seen the perforation nine times in the upper lobe, once in the middle, and eight times in the lower lobe, the anterior, the axillary, and posterior aspect of the lung being affected with nearly equal frequency. The lowest situation in which we have observed the rupture has been the centre of the posterior aspect of the lower lobe.

Whether the communication with the pleura be patent or valvular is a matter of some importance as regards both symptoms and treatment. If the opening be direct and patent, there can be no positive air-pressure within the pleura, since no air can be pent up there. By means of a trocar fitted by tubing to a water-pressure gauge we have ascertained post-mortem the degree of intrapleural pressure in sixteen cases of pneumothorax,[12] and in four out of these the pressure was *nil*. If, on the other hand, the opening be

PLATE V

oblique or valvular, although during inspiration air may enter the pleura, yet the moment the air in the pleura is compressed in expiration the edges of the oblique opening come together, and the air cannot escape at all, or only with difficulty. In twelve cases out of the sixteen above mentioned there was an intrapleural pressure present varying in degree from $1\frac{3}{4}$ to 7 inches of water.

The gas effused into the cavity of the pleura resembles expired air in so far that it contains less oxygen and more carbonic acid than inspired air, but it varies in composition according to the nature of the opening. John Davy,[13] in 1823, was the first to study this question, and in a series of observations on a case of pyo-pneumothorax he found that the carbonic acid reached a maximum of 16 per cent., while the oxygen fell to a minimum of 1·5 per cent. Other analyses have since been made, and notably by Ewald,[14] who, from an examination of eighteen cases, concluded that the nature of the perforation could be gauged from the percentage of carbonic acid present, under 5 per cent. indicating a large perforation communicating freely with a bronchus, 5 to 10 per cent. a valvular aperture, and 10 per cent. or higher a perforation already closed.

These conclusions must not be accepted too exactly, but Ewald's figures are interesting as showing what differences we may expect to find in the composition of the air according as its escape is easy or more or less impeded. Two extreme examples may be quoted, the figures for inspired and expired air being added for comparison:

	Percentage of CO_2.	Percentage of O.	Percentage of N.
Normal inspired air	0·04	20·81	79·15
Pneumothorax with open perforation	1·76	18·93	79·31
Normal expired air	4·38	16·033	79·587
Pneumothorax with closed perforation	17·75	9·95	72·30

In cases of fœtid pyo-pneumothorax sulphuretted hydrogen may be present, sometimes even in considerable quantities (Duncan[15]).

The *first* effect of perforation of the pleura is effusion of

air, and perhaps the escape of some purulent fluid from the ruptured lung into the pleural cavity; the *second* effect is more or less intense pleuritis; and, *thirdly*, if the patient survives, a fluid effusion generally follows. This is usually at first serous, but it becomes opalescent, and may finally assume an opaque and purulent character. For a considerable time, however—possibly for some weeks, or even longer—the sero-purulent appearance is retained, and in tuberculous cases this may be described as the usual appearance of the liquid. Such serous or sero-purulent exudates, when tested, are found sterile; but inoculation into a guinea-pig rarely fails to prove their tuberculous nature, and in not a few cases tubercle bacilli may be detected in the sediment. Occasionally the liquid may be truly purulent from the first, pyogenic micro-organisms having obtained access to the pleural cavity.

The following table shows the nature of the effusion in fifteen cases of tuberculous pneumothorax observed by us, in which the duration could be approximately gauged[9b]:

Duration of Pneumothorax.	Contents of Pleural Cavity.
A few hours	Air only.
30 hours	,,
2 days	,,
2 ,, ...	Air with sero-purulent fluid.
6 ,, ...	Air only.
6 ,, ...	Air with serous fluid.
2 weeks	,, ,,
Some weeks	Air with pus.
At least 5 weeks	,, ,,
2 months	,, ,,
At least 2 months	,, ,,
At least 2½ months	Sero-purulent fluid.
4 months	,, ,,
5 ,, ...	,, ,,
7 ,, ...	Air with pus.
8 ,, ...	,, ,,

Symptoms.—In speaking of the symptoms of pneumothorax, we have especially in mind that most common form due to perforation of the pleura in the course of tuberculous disease. The reader will easily recognise variations from this type. In well-marked cases the principal symptoms are sudden acute pain in the side, followed, or rather attended, by great dyspnœa and shock. The pulse becomes frequent, feeble and small, the respirations relatively more frequent than the pulse, and the voice feeble or suppressed. As a result of the shock, just as in perforation of the intestine,

the temperature of the body may fall. But such a primary fall is rarely a marked feature, and is very often absent. More commonly the temperature at once rises, and in some cases the rise is rapid and marked, forming a noticeable feature of the temperature chart (see Figs. 23 and 24). The patient is often conscious of "something having given way," and feels a peculiar trickling, cold sensation, associated with the pain in the affected side. Occasionally, also, there is

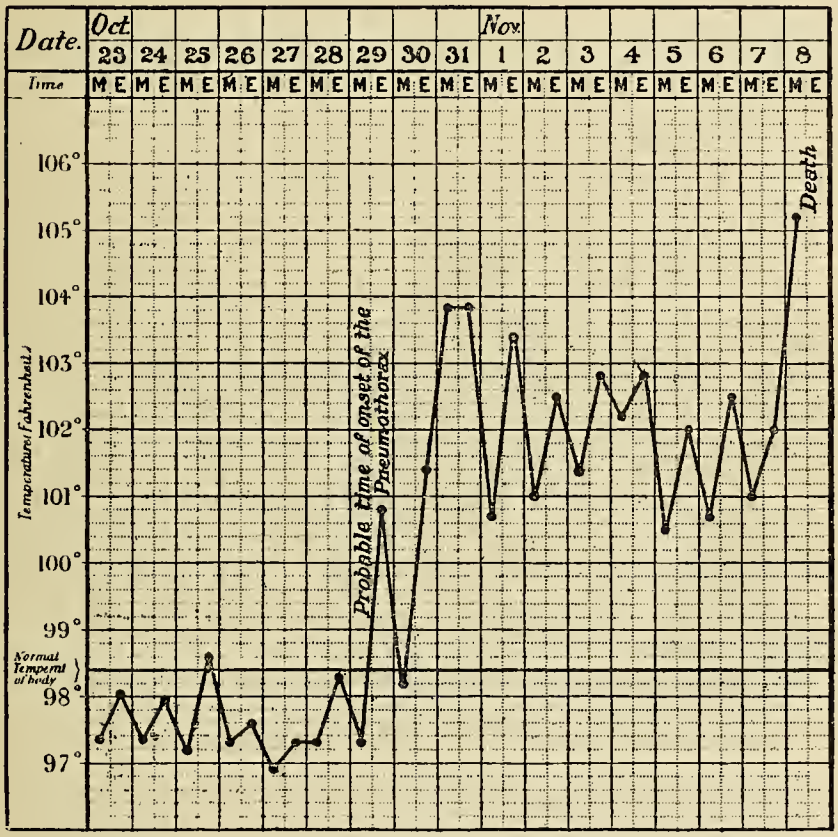


FIG. 23.—CHART ILLUSTRATING THE RAPID RISE OF TEMPERATURE WHICH IS OFTEN ASSOCIATED WITH THE DEVELOPMENT OF PNEUMOTHORAX.

(From a male patient, J. B., aged twenty-one.)

great hyperæsthesia in this region. The patient frequently changes his position: he may sit up or recline, with the head raised, and with an inclination to the sound side, or, again, he may assume the sitting posture with slight inclination forwards, and with the elbows resting upon the knees, and this is the position most commonly chosen.

There is nothing absolutely characteristic about these symptoms except, perhaps, the suddenness with which they may

supervene; but in marked cases the alarmed, anxious, and distressed countenance, the evident urgency of the dyspnœa, usually amounting to orthopnœa, and the small whispering voice, are in themselves strikingly suggestive. All the symptoms may, nevertheless, be most closely simulated in an attack of acute pulmonary congestion supervening upon already advanced tuberculous disease. On the other hand, in not a few cases, and especially those in which the lung affected has

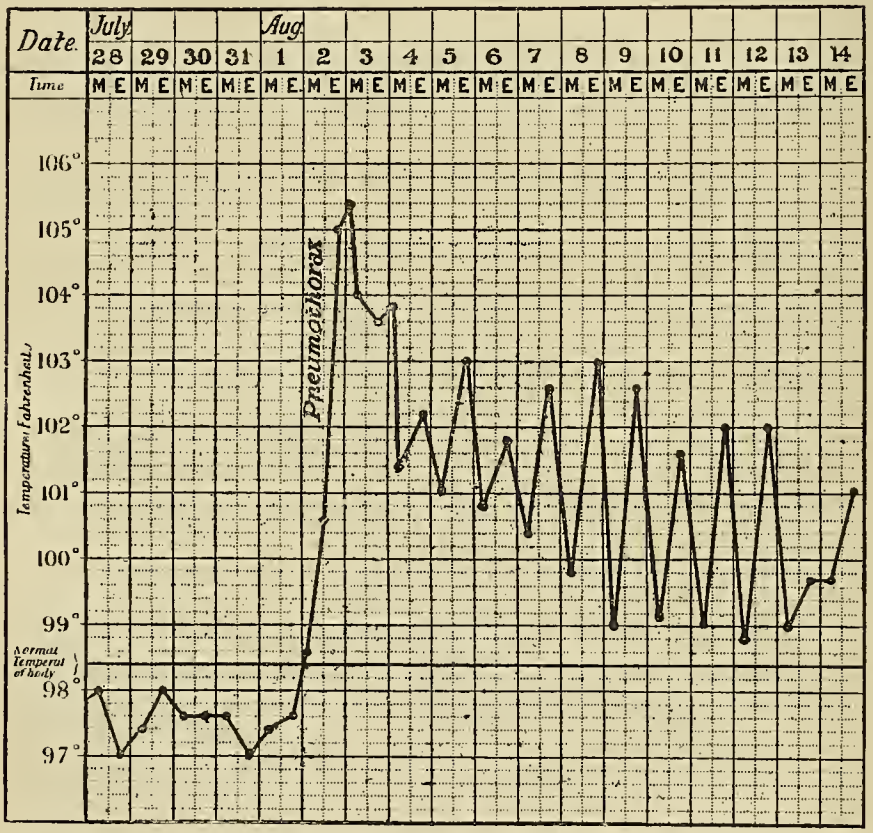


FIG. 24.—CHART ILLUSTRATING THE RAPID RISE OF TEMPERATURE WHICH NOT UNCOMMONLY ACCOMPANIES THE ONSET OF PNEUMOTHORAX.

(From a male patient, W. H., aged nineteen.)

been already extensively destroyed, there may be an almost entire absence of any symptoms to mark the onset of the complication. In such cases disturbed action of the heart may be the chief trouble of which the patient complains.

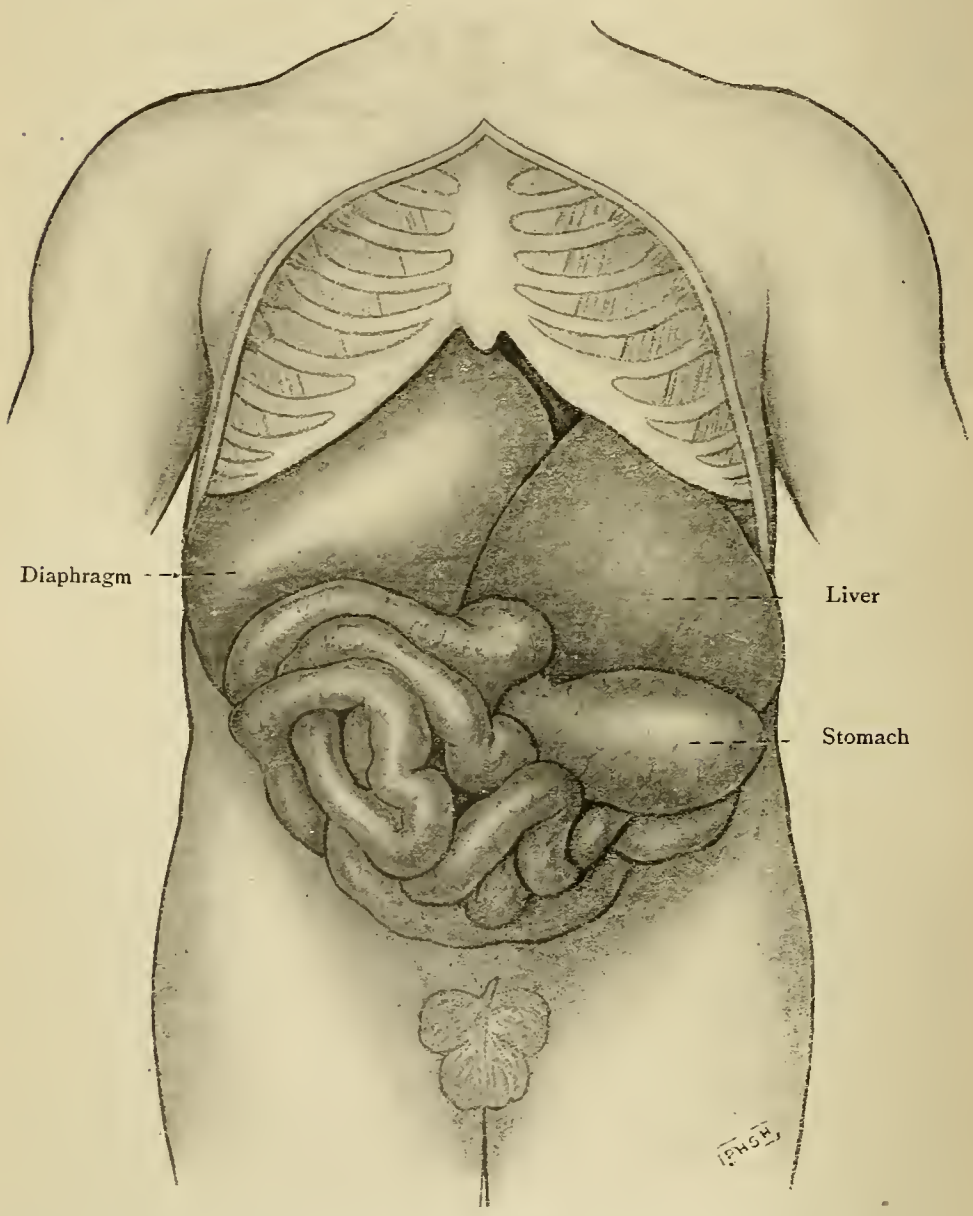
Physical Signs.—The physical signs of pneumothorax are very definite, and can rarely be mistaken for those of any other disease. We will enumerate the chief of them, and dwell more fully upon those which are essentially important.

In a well-marked case the affected side is enlarged, the shoulder raised, the intercostal spaces effaced, and but little or no movement is perceptible with respiration, whilst the opposite side labours with the rapid breathing, its soft parts receding with each inspiration. The heart is displaced towards the sound side, and the abdominal organs are depressed. The percussion note is greatly hyper-resonant or truly tympanitic, where, perhaps, it had before been impaired. Respiratory murmur is either absent or very feeble, and at one or two points more or less distant amphoric breathing may be heard, with metallic whisper and echo on coughing. Pectoriloquy is scarcely ever present. Metallic tinkling may be observed, especially after cough. Vocal fremitus is either absent or much diminished. On applying the stethoscope over one point of the area affected, whilst at another percussion is made with coins or other hard substances, a peculiar ringing sound, the *bruit d'airain* or bell sound (see p. 62), is heard. This is very characteristic of pneumothorax, though occasionally also heard over large pulmonary cavities.

Later, when effusion of fluid has occurred, there is dulness over the lower portion of the affected side, with hyper-resonance above, the distribution of dulness and resonance shifting with the altered position of the patient. The presence of such *movable dulness* is highly suggestive, the level of the fluid in a case of simple pleurisy shifting in a very slight degree, and that only in the earlier stages. On placing the ear against the chest, and giving the patient a somewhat abrupt shake, a splashing sound may be distinctly heard, which is very characteristic of hydro- or pyo-pneumothorax.* This "succussion splash" may be observed in cases of pneumothorax in which no other evidence of fluid has been forthcoming. The explanation is simple. The moment pneumothorax occurs, the diaphragm on the affected side becomes flaccid and more or less concave, being drawn downwards by the weight of the abdominal organs, and in this concavity a certain quantity of fluid may collect without yielding percussion dulness. In cases in which the intrapleural tension

* The directions given in the Hippocratic writings for eliciting this sound are as follows: "Place the patient on a firm seat, and let an assistant hold his arms; then, shaking him by the shoulders, listen on which side the splash is heard" (*De Morbis*, ii., § 47).

FIG. 25.—DRAWING MADE AT THE AUTOPSY OF A CASE OF PYO-PNEUMOTHORAX WITH MARKED INTRAPLEURAL PRESSURE, SHOWING THE DIAPHRAGM EXTENDING DOWN TO THE UMBILICUS, AND FORMING A LARGE ABDOMINAL TUMOUR, GREATLY DISPLACING THE LIVER AND STOMACH. WHEN THE CHEST WAS PUNCTURED, AND THE AIR ALLOWED TO ESCAPE, THE DIAPHRAGM BECAME RELAXED, AND THE TUMOUR DISAPPEARED.



(NOTE.—The drawing was made from near the feet, and the chest is accordingly somewhat foreshortened.)

is considerable this depression of the diaphragm may become extreme. Thus, in the case of pyo-pneumothorax illustrated in Fig. 25 it formed a large sausage-shaped tumour, convex

towards the abdomen, extending nearly to the umbilicus, and dislocating the liver to the left. On puncturing the chest and allowing the air to escape, the diaphragm became relaxed, and the tumour disappeared.

In cases in which the quantity of fluid present is considerable intercostal fluctuation may sometimes be observed. In other cases, on sharply percussing immediately below the line of contact of resonance and dulness, a thrill, significant of fluid vibrations, may be detected.

Such are the signs of pneumothorax, of which those of cardinal importance are the following: (1) hyper-resonance; (2) absent, feeble, or amphoric respiratory sounds; and (3) displacement of the heart. These three signs alone are sufficient to render the diagnosis certain, and their presence can be ascertained by a physical examination, which will not add to the distress of the patient. Let us now consider each of them in greater detail:

1. The degree and extent of the *hyper-resonance* depend upon the quantity and tension of the air that has escaped into the pleural cavity. The note has usually a drum-like quality, which is characteristic; but in cases in which the tension of the air is great the vibration of the chest walls is less free, and the tympanitic note becomes somewhat deadened. The boundaries of hyper-resonance include the sternum, and may extend beyond it towards the healthy side; if the left side be affected, the normal cardiac dulness is altogether effaced. As a rule, the whole lung is collapsed, save perhaps at the summit, where there are frequently some old adhesions. In some cases, however, adhesions are so strong and extensive as to limit the pneumothorax to a small portion only of the pleural cavity—usually the base or the lower anterior and axillary region. In such cases of "partial pneumothorax"—and they are by no means uncommon, nine out of our nineteen cases being of this type[9b]—the hyper-resonance will be limited to the corresponding portion of the chest. As the symptoms are rarely urgent in these patients, the complication may pass unnoticed.

2. The character of the *breath-sounds* varies according to the nature of the opening. Over the greater portion of the affected side the respiratory sounds are as a rule annulled, and where the opening is small and quite valvular no auscultatory

sounds may be detected at any point, although very often, even in these cases, at one spot a faint and distant hollow inspiratory sound may be heard on careful auscultation. In cases, however, in which the opening through the pleura is free, the entry and exit of the air to and from the pleural cavity gives rise to a variety of amphoric breathing—not loud, but peculiarly large and of metallic quality—which can rarely be mistaken. This amphoric breathing is most audible at some one portion of the chest nearest to the seat of perforation. It is commonly best heard at the mammary or upper or lower scapular region, and is conducted more or less distinctly from this point. With a free and patent opening the expiratory portion of the amphoric sound is peculiarly distinct. In these latter cases with free opening, the voice-sounds may be attended with a metallic echo quite peculiar, whereas in the more valvular cases the voice-sounds are not conducted at all. Metallic tinkling is frequently present, and is a useful additional sign—one, however, which may at times be heard very clearly over large pulmonary cavities. Feeble and more or less modified breath-sounds are heard at the apex, where there are still adhesions, and immediately after the occurrence of perforation, friction sounds may develop over portions of lung as yet in contact with the thoracic wall.

3. *Displacement of the Heart.*—M. Gaide[16] was the first to describe displacement of the heart as an important sign of pneumothorax. It is, indeed, a constant and, save in exceptional cases in which the base of the opposite lung is consolidated, an essential sign of perforation of the pleura, and it is singular that it should have escaped the notice of such acute clinical observers as Laennec and Louis. Its occurrence simultaneously with that of the perforation, noticed but not explained by M. Gaide, is a fact that would of itself cast suspicion upon the usual acceptance of the sign as being necessarily one of pressure. The cardiac displacement may be observed within a few minutes of the perforation, and is due, in the first instance, to the sudden removal from the mediastinum of the elastic traction of the lung which has collapsed, and the consequent unopposed traction upon it of the other lung. If the opposite lung be not solidified, the heart may from this cause alone be carried beyond the median

PLATE VI

Radiogram of Chest in a Case of Right-Sided Pyo-Pneumothorax.
(Taken by Dr. Stanley Melville.)

To face p. 14

PYO-PNEUMOTHORAX

The radiogram shows the appearances met with in a case of right-sided pyo-pneumothorax, the radiogram being taken with the patient in the standing position.

The heart and mediastinum are drawn somewhat to the left, and the left lung shows evidence of tuberculous infiltration. The deep shadow of the fluid, with horizontal upper limit, is well seen on the right side, together with the bright air-containing space above. The right lung is collapsed along the vertebral column, except at the apex.

From a man aged twenty-six, who suffered from pulmonary tuberculosis.

PLATE VI

line. Thus, we have recorded two cases, and have seen several others, in which the heart was displaced to the right of the sternum, yet in which, as proved post-mortem by the manometer, no intrathoracic pressure existed.[17]

On a screen examination by the X-rays the affected side is seen to be brighter or more transradiant than the sound side, owing to there being now less tissue to oppose the passage of the rays (see Plate VI.). The shadow of the collapsed lung may be observed by the side of the vertebral column, and the displacement of the heart, already detected by physical examination, will be confirmed. If fluid be present as well as air, the appearances seen are striking. In the erect posture the upper portion of the chest, where the air is now collected, will show the increased translucency characteristic of pneumothorax, the lower portion yielding the dark shadow of fluid. The line of demarcation between the two is sharp and clear. Further, the level of the fluid, unlike that of a simple pleural effusion, is absolutely horizontal—a water-level in any position which the patient may assume. This fact, when observed, is of great importance, and proves the presence of a cavity within the chest containing air and fluid. The upper level of the fluid, whether serum or pus, is rarely still, and this is especially the case if the disease be left-sided, the surface showing continual rippling movements conveyed from the heart, and presenting a very striking picture. On shaking the patient, the fluid is seen to dash against the sides of the pleural cavity and to rebound, forming waves, which accurately explain the mechanism of the succussion splash.

Course and Prognosis.—In the majority of instances pneumothorax occurs towards the close of the disease, when the patient is already dying of extensive pulmonary lesions. It is as a rule, therefore, of grave augury, although the practice of early evacuating the air from the distended pleura has considerably diminished the immediate danger of the complication.[18]

Should the patient survive this primary danger, the heart and circulation gradually become accustomed to the altered conditions, and in the most favourable cases the air, after a few weeks, becomes absorbed, the lung re-expands, and the heart returns to its normal position. Such an event

is not uncommon when pneumothorax occurs in persons apparently healthy, or in whom the lesions of pulmonary tuberculosis are so slight as to give no physical signs. But in cases of manifest phthisis such recovery, though we have known instances of it, is rare. Fluid, usually of a serous or sero-purulent nature, generally becomes effused. This may after some weeks be reabsorbed, the air at the same time disappearing. More commonly it happens that the fluid remains, and if the perforation in the pleura has healed, and the lung is unable to expand, the case becomes converted into one of chronic hydrothorax. This latter termination is, however, exceptional. As a rule, when a hydro-pneumothorax has been established, the condition remains unchanged for months, or even in exceptional cases for years,* until the patient dies from the spread of the primary disease, or more rarely from the sudden onset of acute fœtid pleurisy, putrefactive organisms having found access to the pleura. We have more than once met with cases in which the occurrence of pneumothorax has seemed to arrest the activity of the disease in the lung affected, and to prolong life. Of fifty-eight cases of pneumothorax collected from the post-mortem records of the Brompton Hospital, the greatest durations of life were twelve, eight, seven, five, four and a half, and four months in six cases respectively. The shortest durations were ten minutes, fifteen minutes, and six hours.

We have been speaking so far of the prognosis of pneumothorax when secondary to pulmonary tuberculosis. In other conditions the outlook must vary with the cause. In cases of gangrene of the lung, the rupture of septic infarcts, and bronchiectasis the result is almost always fatal, partly on account of the primary disease and partly from the severe septic inflammation originated in the pleura. In cases of emphysema, on the other hand, and of empyema bursting into the lung, the prognosis is more favourable, provided that in the latter case the pus is freely evacuated by surgical measures.

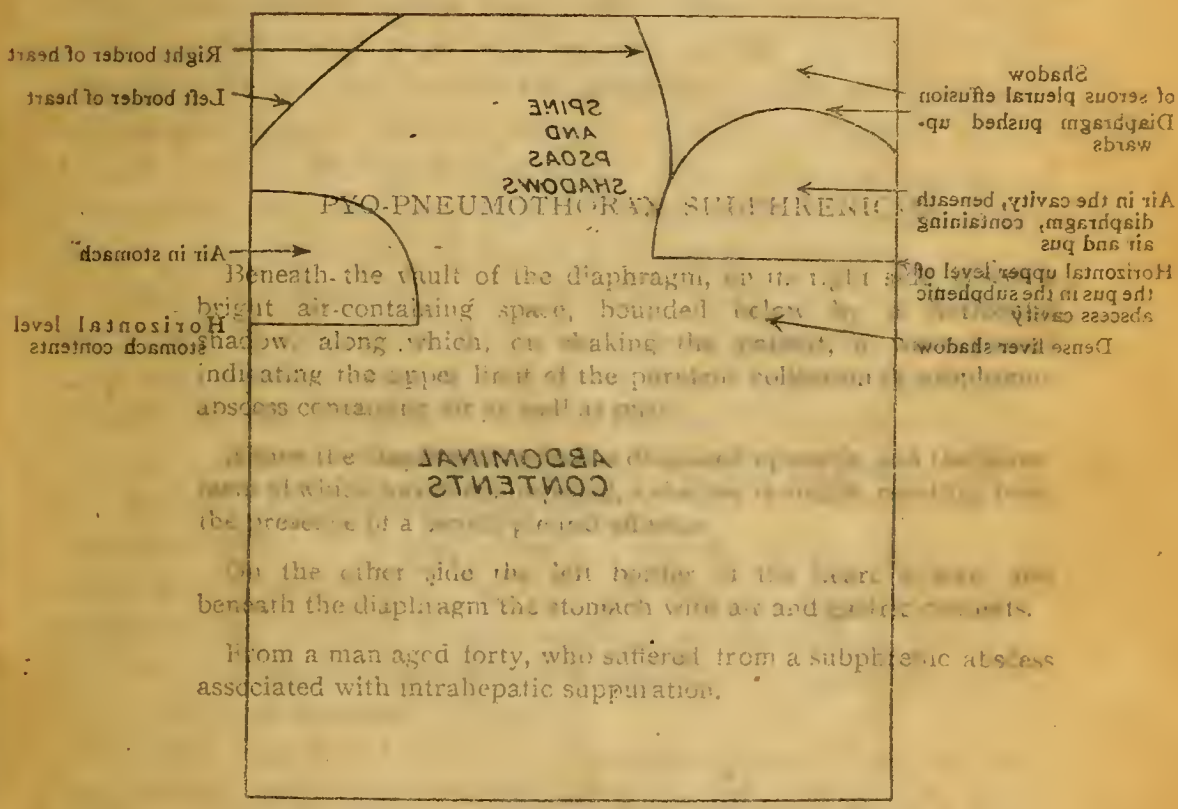
Diagnosis.—There is as a rule but little difficulty in distinguishing a case of pneumothorax, in which the effusion of

* The experience of Dr. Morse,[19] who records six instances of recovery out of fourteen cases of tuberculous hydro-pneumothorax with serous effusion, is most exceptional.

PLATE VII

Radiogram of a Case of
Pyo-Pneumothorax Subphrenicus, viewed from the Front.
(Taken by Dr. Hugh Walsham.)

To face p. 149.



PYO-PNEUMOTHORAX SUBPHRENIC.

Beneath the vault of the diaphragm, on the right side, bright air-containing space, bounded below by a [illegible] shadow, along which, on shaking the patient, [illegible] indicating the upper limit of the purulent collection [illegible] abscess containing air as well as pus.

[illegible] the [illegible] the presence of a [illegible] pleural effusion.

On the other side the left border of the heart [illegible] beneath the diaphragm the stomach with air and gastric contents.

From a man aged forty, who suffered from a subphrenic abscess associated with intrahepatic suppuration.

PLATE VII

air is extensive, from any other morbid state. *Emphysema* is the only other affection in which we obtain hyper-resonance and enfeebled breathing combined. But in emphysema the disease affects both lungs, the note lacks the drum-like quality found in pneumothorax, the respiration is never quite suppressed, it is not amphoric in quality, and the heart is not laterally displaced.

It is sometimes very difficult to distinguish between a localised pneumothorax and a large thin-walled *pulmonary cavity*. Such cavities may yield almost tympanitic resonance and metallic tinkling, whilst the bell sound, movable dulness, and succussion splash may all, though rarely, be elicited, as in a case which we have recorded.[20] But localised pneumothorax is most commonly situated at the lower portion of the thorax, and in this situation such a large pulmonary cavity as could be confounded with it is of most rare occurrence, unless it be continuous from the apex downwards, in which case the heart's beat would be felt on the *affected* side. Except for the position of the heart, the X-ray appearances of *total excavation of the lung* may exactly resemble those of a hydro- or pyo-pneumothorax.[21]

Occasionally a diaphragmatic hernia, with escape of stomach and colon into the left side of the chest, and with displacement of heart to the right, may simulate a pneumothorax. But here a bismuth meal and X-ray examination will reveal the true nature of the case.

Another source of error is the occurrence of an abscess beneath the diaphragm containing both air and fluid, a condition originally described by Leyden[22] under the name *pyo-pneumothorax subphrenicus*. This is most often secondary to perforation of a gastric or duodenal ulcer. The abdominal symptoms in the preceding history of the case, the absence of cough, expectoration, and of pulmonary signs in the upper portions of the chest, together with the comparatively slight cardiac displacement, are points which should help to direct diagnosis aright. An X-ray examination, provided the patient's condition permit, gives valuable confirmatory evidence, revealing the fixed condition and upward displacement of the diaphragm, and beneath it the presence of a cavity containing air and fluid (see Plate VII.).

But the cases which we have seen most often mistaken for

pneumothorax have been those of advanced phthisis, in which *acute congestion* has rapidly supervened at the base of the comparatively sound lung. Pain limits the movements and lessens the sounds over the newly affected part; there is considerable high-pitched resonance on percussion; and the symptoms, setting in suddenly and acutely, may be precisely those of pneumothorax. But on careful auscultation breath-sound and rhonchus can be heard; the heart is not displaced, nor is the percussion note truly tympanitic.

Sometimes, also, at first sight the dyspnœa of asthma resembles that of pneumothorax, and with general hyper-resonance we may have in asthma an absence of respiration over portions of the chest. But the portions of lung so affected will vary in position, perhaps, even whilst we are listening, and the general wheezing râles elsewhere present, together with the history of the case and the effect of treatment, will prevent any real difficulty in diagnosis. Again, we have seen more than one case of *hysterical dyspnœa* suggesting pneumothorax, but the expression of countenance cannot be simulated, and a moderately careful physical examination will lead to a right conclusion.

The diagnosis of pyo-pneumothorax from simple empyema is not difficult, the succussion splash and the marked shifting of the dulness and resonance with change of posture, as well as the X-ray appearances, being quite characteristic of the former. Nevertheless, we must remember that certain cases of apparently pure empyema have their origin in perforation of the lung, the rupture having closed and the air undergone complete absorption.

But the diagnosis in pneumothorax does not consist merely in separating it from other diseases, but also in distinguishing the kind of perforation that has taken place and the probable existence or not of air-pressure within the thorax. The discovery of amphoric (to-and-fro) breathing strongly suggests that the opening is a free one, admitting the ready passage of air both ways, and that, consequently, no air-pressure is present. On the other hand, the complete absence of all breath-sounds, with increasingly urgent dyspnœa, distended side, and greatly displaced and oppressed heart, are equally significant of a valvular opening and of increasing intra-thoracic pressure.

Treatment.—Life is threatened on the occurrence of pneumothorax by *shock*, *asphyxia*, and *exhaustion*, and these are the special indications for treatment. The shock, which is due to the sudden lesion of a vital organ and to the sudden dislocation and impeded action of the heart, must be treated by the administration of a stimulant, but above all things by an opiate. Opium is most valuable in calming the nervous system and in lessening the sense of dyspnœa. A dose of morphia may be given subcutaneously, and the treatment continued in the form of a pill of opium with camphor. Increasing pressure within the chest, if present, must be treated by the timely introduction of a fine trocar. For this purpose a Southey's trocar with fine rubber tube attached may be employed. The tube should open below into boracic lotion to prevent the entry of air into the chest during inspiration, but in emergency any fine instrument may be used. This trifling operation gives great and, curiously, sometimes lasting relief. If necessary, it may be repeated. After the excess of air has been removed, the side may be strapped so as to control inspiratory movement. A broad band of strapping firmly applied over the lower ribs, and reaching some two or three inches beyond the middle line in front and behind, is sufficient to attain this end. Positive pressure is brought about within the pleura by the thoracic wall on the affected side becoming expanded to the position of extreme inspiration, and then recoiling upon the air pent up in the pleura. By diminishing the inspiratory movements we may hope to prevent this excessive accumulation of air, with all its attendant consequences.

As a rule pneumothorax occurs in persons already reduced in flesh and blood by previous illness. If the accident should occur at an earlier period of the disease, the venous engorgement, lividity, and general circulatory distress resulting from the embarrassment and dilatation of the right side of the heart, will be correspondingly marked. In such cases free dry-cupping will give great relief. The portal system being the great reservoir for retarded blood, an occasional saline aperient is of value in pneumothorax, and also serves to correct the constipating effects of opium, the administration of which in repeated small doses is on other grounds desirable for the first few days.

When *effusion* has occurred, we must not be in too great a hurry to withdraw it, provided it be of the usual serous or sero-purulent variety, since a certain delay will assist in closing the rupture in the pleural membrane. But should pressure symptoms ensue, we may remove a portion, care being taken not to produce a negative pressure in the pleura, lest the rupture recently healed be reopened. The syphon may, therefore, be employed, or, if the aspirator be used, the fluid must be allowed to flow with as little suction as possible.

If the fluid returns, a second paracentesis may be performed; but, should this also fail, it is better as a rule to suspend active treatment. The patient suffers little, and, provided the disease in the other lung be fairly quiescent, he may live for many months, or occasionally for some years, in that condition, dying at length from the spread of tubercle in the opposite lung, or from the invasion of the larynx and more distant parts.

Should the patient, however, complain of the weight in the chest caused by the effused fluid, relief may be given, and possibly some expansion of the lung effected, by paracentesis followed by *oxygen replacement*,[23] as described when speaking of cases of chronic pleurisy (see p. 114). But we must remember that in cases of pneumothorax any traction exerted upon the lung must be gentle lest the perforation which has now closed should reopen, with the attendant danger of septic infection.

In a case of *pyo-pneumothorax* when the fluid is distinctly purulent and contains pyogenic micro-organisms, and pyrexia is present, paracentesis should be performed and repeated if necessary, but if the patient continues to lose ground, incision and evacuation of the pus, with subsequent drainage, is our only course, provided the general condition does not forbid. In certain cases thus treated the perforation in the pleura has closed, and the lung has re-expanded, though a fistula may persist.[24] Should the expansion of the organ be incomplete, and the chest not fall in sufficiently, later on a modified Estlander's operation may be required. We have known a remarkable case in which this was successfully performed. Each case must, however, be separately considered, and treated on its own merits. If there is little or no fever, and the patient's condition be satisfactory, it will be wiser to leave well alone,

to perform paracentesis from time to time when required, but not to proceed to more drastic surgical measures. How long cases of pyo-pneumothorax not operated upon may continue to live is well exemplified by the history of the following patient:

Mr. T., aged twenty-nine, was first seen in 1902. His father had suffered from "weak lungs," and died early, but the family history was otherwise satisfactory. He was delicate as a child, but became robust at sixteen, and excelled at sports, being accustomed to contend in fifty-mile bicycle races with success. In 1895 he was passed as medically sound for the public service, but for family reasons he entered upon a commercial career in London. In 1897 he married, and four children were born, one of whom died at birth, and one of tuberculous meningitis at fifteen months.

In 1901 he contracted repeated "colds," and in December of that year he developed early tuberculous trouble at the left apex, with tubercle bacilli in the sputum, and, by the advice of his family physician and a consultant, went to Torquay, where he improved considerably for a time.

On May 9, 1902, he was sent to Sir R. Douglas Powell by Dr. Edwin Smith, of Tooting. Consolidation of the upper third of the left lung was found, but the constitutional symptoms were mainly in abeyance. The alternatives of his remaining in town with precautions and of his taking a voyage were considered and negatived, and treatment at a sanatorium for at least three or four months was strongly advocated, with a hopeful prognosis. Some creosote was prescribed.

In September of that year he returned from a four-months course of treatment at a Hampshire sanatorium, having gained 1 stone in weight (9 stone 2 pounds), with a normal temperature and only slight morning cough. He could walk ten or fourteen miles a day. Although the physical signs at the left apex remained, with some coarse crackles, the right lung had expanded across the sternum. He went to Bournemouth, where he bought a house, and remained with his wife and family. In January, 1903, his weight was 9 stone 6 pounds, the disease quiescent, with some fibroid changes. Temperature normal.

In June, 1904, he reported that he had early in the year had some relapse of symptoms. His child had died from tuberculous meningitis, and he had suffered from pleurisy, with loss of weight. For the last six months, however, his temperature had been normal, and his present weight was 9 stone 4 pounds.

In May, 1905, he developed pneumothorax on the left side below the fifth rib in front and behind, followed later by fluid effusion. He was exceedingly ill for some time, and at the end of four months was only just able to stand. He then again improved rapidly, and after nine months reported himself as better than before the pneumo-

thorax. His cough had disappeared, he had gained weight, and the temperature was normal. The signs of pneumothorax, however, persisted. His letter, written on July 9, 1906, contains the following interesting passage: "Of the thirteen patients of a group taken when I was at the sanatorium (in 1902), nine are dead. Of the remaining four I am one; of two I am doubtful, and *one* is really soundly cured, and he is the careless one who plays hockey in Ireland. . . . I have just noticed we were thirteen! . . . I have been through more than any of those who died, but I think it has much to do with will-power, and not allowing oneself to worry." In regard to this psychical aspect of the matter, Mr. T. deprecates the practice of enjoining patients to take their temperature three times a day, adding: "When it is up, they worry until it goes higher. I went to 105·2°, but I did not mind. A sensitive girl would have died of fright. I am a layman, I know, but I feel that mind is half the battle one way or another."

In June, 1907, Mr. T. was seen again in consultation with Dr. Hyla Greves, and he was found still to present all the physical signs of pyo-pneumothorax, communicating with and discharging through the lung. The question of opening up the pleura, with removal of some of the ribs, with the hope of closing the cavity, was carefully considered, and was reluctantly abandoned as a measure that would probably have had an immediately fatal result. He was advised to evacuate the pleural cavity as far as possible by natural means and at stated intervals. With his usual determination he set to work to carry out directions by a process of "tilting-up," as he expresses it, "lying on a lounge-chair with head on the floor and legs up the back of the chair," thus inducing cough and expectoration.

In February, 1909, Mr. T. wrote that since June, 1907, he had "tilted up" twice and later three times a week, and had brought up altogether about 10½ gallons of pus, carefully measured, but that at the date of writing he expectorated only about ½ pint each time. Recently there had been intervals of perhaps a week without expectoration, "the safety-valve having struck work," as he describes it, during which time he became "depressed, weak, and austere. The block then gives way, and at the next attempt at evacuation there is a sudden rush, and 1½ pints of pus may be brought up." In a later letter, in May, 1910, he states that he is much better, expectorating only about an eggcupful every day, though the total amount evacuated by the tilting process had now reached 15 gallons. On October 25, 1910, Dr. Hyla Greves writes: "At the present time Mr. T.'s condition is very fairly good, considering all he has gone through. The left side of the chest has fallen in greatly, and the pneumothorax cavity correspondingly diminished in size, the amount of expectoration discharged by 'tilting up' being comparatively small. The right lung shows very few indications of disease, but there are a few moist sounds in the upper lobe. Some months ago he had a

good deal of abdominal pain and discomfort, and I feared the possibility of tuberculous disease of his intestines, but careful dieting and having his teeth put into proper order has apparently got rid of this condition. He is able to take a moderate amount of walking exercise daily."

The patient continued under Dr. Greve's skilful and inspiriting care and kept fairly well, but the disease in the right lung made progress, and signs of a cavity developed in the upper lobe. On April 22, 1912, Mr. T. had his first attack of hæmoptysis, the blood probably coming from the right lung. The hæmorrhage recurred on several occasions and greatly reduced his strength. He died in June, 1912.

This case is in some respects a remarkable one of endurance and will-power in contending against a disease which would certainly have sooner proved fatal in a less sanguine and determined man, and on this account appears worthy of record. The death-rate incidentally mentioned amongst his sanatorium contemporaries is interesting.

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CHAPTER X

HÆMOTHORAX—GUNSHOT WOUNDS OF THE CHEST

By the term Hæmothorax is meant the effusion of pure blood into the pleural cavity, the condition being distinguished from hæmorrhagic pleurisy, in which the inflammatory exudation is merely tinged with blood.

Hæmothorax may be secondary to disease, or result from injury, such as fractured ribs, but is most commonly a sequel of gunshot wounds of the thorax, and as such has been of frequent occurrence during the recent war.

The amount of blood poured out varies from a few ounces to several pints, according to the extent of the wound and the source of the hæmorrhage, whether coming from lung, aorta, intercostal or internal mammary artery.

As Colonel Elliott and Major Henry[3] have shown, the blood rapidly clots, but, owing to the cardiac and respiratory movements, the clot is not, as a rule, massive, and the fibrin is deposited as a layer upon the pleural surfaces. If the fluid be removed from the chest and tested for fibrinogen none will be found, showing that clotting has, in fact, occurred, and that the fluid is really defibrinated blood. As a result of the irritating action of the blood a low degree of pleurisy follows, and serous fluid is poured forth, mixing with and diluting the hæmothorax.

Hæmothorax in Civil Life.

In civil life hæmothorax is not common, and is then perhaps most often the result of fracture of rib with lacerated pleura. Another cause is the rupture or leakage of an aortic aneurism into the pleural cavity. Ulcerative erosion penetrating the aorta, intrathoracic veins, or intercostal arteries has also been described as a rare precursor of this condition (Watson). In

malignant or tuberculous disease hæmorrhagic pleurisy, as we have already seen, is not uncommon; but definite hæmorrhage into the pleura is rare. In scurvy, purpura, and the malignant forms of the specific fevers, the fluid effused into the pleura may be somewhat blood-stained, but here also a true hæmothorax is exceptional.

It sometimes happens that with the blood air makes its way into the pleural cavity, and a *hæmopneumothorax* results, an uncommon condition in civil practice, of which but few cases are recorded. In an interesting paper Dr. Newton Pitt[1] described a case due to the rupture of an emphysematous bulla, and referred to three other cases from the post-mortem records of Guy's Hospital. In two of the latter the condition resulted from a laceration of the lung (without fracture of the ribs) following a crush, and in the third from the rupture of a phthisical cavity through the pleura, this event being followed shortly afterwards by hæmorrhage from an ulcerated vessel in the cavity wall, leading both to hæmoptysis and to the effusion of blood within the pleura.

Symptoms.—The symptoms of hæmothorax unassociated with wounds do not differ materially from those of other pleuritic effusions, unless the hæmorrhage be profuse, in which case restlessness and distress, a rapid and irregular pulse, and other signs of internal hæmorrhage are superadded. The physical signs are indistinguishable from those of other exudations into the pleura, and except when caused by injury, the condition is rarely suspected until exploratory puncture reveals its nature. The rapidity with which the fluid collects might in some cases, however, suggest a suspicion as to the true state of affairs.

Treatment.—If the hæmothorax result from the leaking of an aortic aneurism, clearly we should not interfere unless great displacement of heart and increasing dyspnœa render paracentesis imperative, since the pressure of the effusion will tend to check the further flow of blood. In other cases, if the effusion be large, paracentesis may be employed, earlier recovery having been observed during the war from the withdrawal of the fluid. Each case must be judged upon its merits, it being borne in mind that, if the primary disease permit recovery, the blood will be gradually absorbed by natural means, though this process may be slow. Should signs of

suppurative pleurisy supervene from infection of the pleura, surgical treatment must not be delayed.

Hæmothorax following Chest Wounds.

As we have stated, hæmothorax has been of frequent occurrence during the war, and it may be said to be the common sequel of a gunshot wound of the chest. In such cases the blood comes, as a rule, from the injured lung, more rarely from a damaged intercostal or internal mammary artery. It is not uncommon for a small quantity of air to be present also in the pleura during the early stages, and for the case to be strictly a hæmopneumothorax, but the quantity of air is rarely large, and it is in most cases rapidly absorbed.

Symptoms and Physical Signs.—A certain degree of shock always follows a wound of the chest. In grave cases it is severe, and it is estimated that, taking all chest wounds together, an early mortality of from 10 to 15 per cent. results from shock and primary hæmorrhage.[6] In large open wounds exposing the pleural cavity, so that air is sucked in at each inspiration, so called *Traumatopnœa*, shock and respiratory distress prove fatal unless relieved by surgical treatment; but in simple through-and-through wounds of the chest, or those in which a small missile is retained, these symptoms soon abate, and the case becomes one of hæmothorax, though we must add that an effusion of blood into the pleural cavity does not necessarily occur in all such cases. Hæmoptysis is of frequent occurrence, and may recur for several days, but unless it arises from damage to a large vessel, when it may be rapidly fatal, it is rarely troublesome.

The physical signs of hæmothorax resulting from a penetrating wound of the chest are sometimes those of pleural effusion—namely, impaired note, diminished vocal fremitus, breath-sounds and voice-sounds, with displacement of heart towards the sound side, and if the effusion be of sufficient extent, enlargement of the affected side. As Sir John Rose Bradford[5] has, however, pointed out, in many cases the signs are different, and the affected side, though dull over the effusion, is retracted and almost motionless, and the diaphragm elevated. Bronchial and even cavernous breath-sounds with bronchophony and ægophony are heard over the area of fluid, and above this level, marked skodaic resonance. These

signs have often led to the mistaken diagnosis of traumatic pneumonia, but the heart's apex-beat, be it noted, is usually displaced towards the healthy side. It would appear that these peculiar signs are the result of an underlying *massive collapse of the lung*,[4 and 10] a condition which will be discussed more fully in Chapter XX.

Course.—The blood poured out in a case of gunshot wound of the chest rapidly reaches its maximum, owing partly to the collapse of the lung, which checks the outflow, and partly to the deposition of fibrin, which soon occurs, and which tends, by forming adhesions, to circumscribe the effusion. There is still some dyspnœa on recovering from shock, but it is not urgent, nor is cough as a rule a troublesome symptom. Moderate pyrexia may persist for some little time. *Provided, however, the hæmothorax remains sterile*, these symptoms gradually subside, and the fluid is slowly absorbed.

In some 25 per cent. of chest wounds *sepsis* unfortunately occurs,[6] a complication twice as frequent when the wound has been caused by shell fragment as by machine-gun or rifle bullet.[6] The infecting organisms are attached to the missile, or are introduced with fragments of clothing, or come from within from the lacerated lung. Of 101 cases of septic hæmothorax investigated by Colonel Elliott and Major Henry,[3] in rather less than 20 per cent. the organisms isolated were the pneumococcus, Pfeiffer's bacillus, and the *M. tetragenus*, coming presumably from the respiratory tract: in the remainder, streptococci, staphylococci, and anaerobic gas-forming bacilli, of which *B. perfringens* was the most frequent, were found, gas-forming organisms occurring in nearly half of all the infected cases.

Continued pyrexia, restlessness, a rapid pulse, and in some cases a furred tongue, will suggest the occurrence of sepsis; but to make certain the fluid removed by exploratory puncture must be examined bacteriologically, both by staining and culture. Colonel Elliott and Major Henry[3] found that in 90 per cent. of the infected cases organisms could be detected in films stained with methylene blue and by Gram's method, and that the finding of a stout Gram-staining anthrax-like bacillus was very suggestive evidence of anaerobic infection. Cultures should, however, be made in all cases.

In patients infected with anaerobic organisms the amount of

gas produced within the chest may be considerable, and the pressure as much as + 15 to + 20 cm. of water.[3] Such cases simulate closely, and have been mistaken for *simple hæmopneumothorax* under the impression that the air had entered the pleura from the pulmonary wound. Exploratory puncture, and the withdrawal of fluid, often foul-smelling and containing infecting organisms, will decide the diagnosis.

The occurrence of sepsis is a grave complication, for whereas cases of simple hæmothorax, which fortunately number three-quarters of the whole, gradually make a complete recovery, of those in which sepsis occurs nearly half die.[6 and 14]

Treatment.—(*a*) *Without Operation.*—If the hæmothorax is small, reaching not higher than the angle of the scapula, it may be left alone. The fluid is absorbed and the lung expands, and the patient is in most cases fit for duty in two to three months.

The same policy of non-interference may be followed in regard to larger effusions, and it is astonishing how quickly in some cases the blood is absorbed; but statistics show that recovery is hastened by aspiration. This had previously not been regarded as correct treatment owing to the fear that secondary hæmorrhage might result if the compressing action of the fluid were withdrawn. Experience in the Great War has, however, shown that this fear is unfounded, and if the effusion is a large one it is wise to aspirate. As much of the fluid as possible should be removed, but complete evacuation of the fluid is not necessary, since what is left will be dealt with easily by the pleura. Captain Fortescue-Brickdale[12] has shown that of cases of hæmothorax admitted to the Centre for Gunshot Wounds of the Chest at Southmead, Bristol, 42 per cent. of the *large* non-aspirated cases recovered completely in two months or less, and 84 per cent. of those which had been aspirated, showing the value of aspiration in hastening recovery. In patients with a small hæmothorax, about 90 per cent. recovered in two months or less, whether aspirated or not.

There remains to consider certain rare cases of non-infected hæmothorax, in which the blood-clot is a massive one, and the fibrin not whipped out and deposited as usual on the pleura. In such cases aspiration fails, or is only successful to a limited extent, and if the hæmothorax be a large one it has been

suggested that the pleura should be opened in the manner to be described later, the clot evacuated, and the chest again closed. In view of the fact that, provided sepsis does not supervene, the patient will, with the aid of pulmonary exercises and movements probably in the end make a complete recovery, this course is hardly to be recommended as a routine measure.

(*b*) *With Operation.*—We have so far concerned ourselves with the treatment of cases of hæmothorax following chest wounds which remain uninfected, and they number about 75 per cent. of the whole. If properly treated the prognosis in these cases, as we have seen, is good. Should the hæmothorax become infected, rib resection and drainage must be undertaken, but the outlook in such cases is gloomy, Colonel Elliott [6 and 14] having shown that nearly half the patients succumb, and of the survivors one-third are more or less disabled, and eventually invalided from the army.

We are, therefore, faced with the question whether by surgical means we cannot *prevent infection*, and thus avoid the mortality associated with this complication. Any operation for this purpose must be undertaken early, as soon as the patient has recovered from the initial shock, and will involve excision of the external wounds of the soft parts, the removal of splinters of ribs and, if necessary, resection of broken portions, the cleansing of the pleural cavity from blood and clot, the extraction where possible of the missile, the excision or cleansing of the lung wound, and, lastly, the closing of the chest. To effect these objects a portion of rib is excised, when, with the help of a retractor, sufficient space will be obtained for the introduction of the hand within the thorax and the carrying out of the necessary manipulations. Into the details of the operation we do not propose to enter, and must refer the reader to the writings of Colonel Gask, and others, references to which will be found at the end of this chapter. We may add, however, that severe though the operation sounds, it is, in fact, attended with less shock and a smaller fall in blood-pressure than is often associated with abdominal operations.[9 and 15]

Before the War such operations were rarely undertaken, from the belief that unless some form of pressure chamber, such as those devised by Sauerbruch and Willy Meyer, was

employed to prevent pneumothorax and collapse of the lung when the chest was opened, sudden death would follow. Recent experience has shown that this fear is groundless, and it is estimated that during the Passchendaele fighting in the summer and autumn of 1917, 38 per cent. of the chest wounds were operated upon on the lines indicated above.[14] It is clear, however, that as sepsis occurs in only a quarter of the cases, and as recovery in uninfected hæmothorax may be anticipated, a rigid selection must be made, those chosen for operation being the cases in which, from the nature of the wound and of the missile, sepsis is most likely to supervene. In the opinion of Colonel Gask, the more important indications for operation are: a ragged external wound; compound fracture of the ribs; continued bleeding; an open wound leading into the pleural cavity with insuction of air at each inspiration; the retention of a large foreign body; and pain, out of proportion to the injury, and often due to indriven splinters of bone, which so often lead to sepsis.

We may say in conclusion that there can be no doubt that in cases of gross injury, such as those in which the chest is open, or in which a large foreign body is retained, or those which contain large indriven fragments of bone or clothing, operation is urgently demanded and often life-saving. No patient, on the other hand, with a small and clean-cut wound, in whom the foreign body is of small dimensions and of smooth surface, and whose general condition is good, should be submitted to operation. Between these two groups there must be many intermediate cases in which treatment must depend upon a careful consideration of all the surrounding circumstances. In certain of these patients, provided the services of a surgeon experienced in chest work are available and the conditions for operation satisfactory, a complete operation, as sketched out above, may be sanctioned, for there can be little doubt that in the hands of those who have especially devoted themselves to chest work such operations have led to a saving of life, though the results from the army as a whole, quoted by Colonel Elliott,[14] are less encouraging. If, on the other hand, the indications for operation are not so clear, and the surrounding circumstances less favourable, then it will be better merely to excise the external wound, to remove fractured portions of ribs, and then to close the wound, treating the

hæmothorax, if necessary, by aspiration, and, should sepsis arise, to resect and drain the pleural cavity.

We may add that we have treated the subject dealt with in this chapter at some length, because there can be no doubt that the lessons learnt in the war in regard to the surgery of the chest will be soon applied to the maladies and injuries of civil life. It is essential, therefore, that both physicians and surgeons should be well acquainted with the subject. As an illustration we may quote the following case, which recently came under our notice:

The patient, a dustman, aged forty-eight, during the course of his work ran a needle into his back. This disappeared within the chest, and caused him at first but little inconvenience, but a few days later (on November 6, 1919) he was seized with sudden pain in the right side, and came to St. Bartholomew's Hospital, where he was seen by one of us. On examination he was found to have surgical emphysema over the lower part of the right lung, with signs suggestive of the presence of a limited quantity of air in the pleural cavity. The screen examination, however, revealed nothing abnormal. He was admitted under Sir Archibald Garrod, and an X-ray plate showed later the presence of a needle in the lower portion of the right chest. On November 14 Mr. Gask operated, the anæsthetic used being chloroform and oxygen. Four inches of the sixth rib in the right axillary and anterior regions were excised and the chest fully opened. Air was found to be present in the pleural cavity, and in the lower portion of the semi-collapsed lung the needle was at once seen, half embedded, half exposed. Except for a little lymph round the site of the puncture, the lung appeared healthy. The needle was extracted and the wound closed. The operation was followed by a small collection of clear serous fluid at the right base and a low degree of pyrexia lasting twelve days, but recovery was otherwise uneventful.

This case shows the value of such methods of surgical treatment, for there can be little doubt that had the case been left alone, suppuration, with its accompanying dangers, would have ensued.

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CHAPTER XI

CHYLOTHORAX

THE occurrence of effusions into the serous cavities which have an appearance resembling that of milk, but which are non-purulent in nature, has been known since the year 1633, when Bartolet[1] described a pleural effusion of this character. It is only in comparatively recent years, however, that the subject has attracted much attention. Such collections are met with most often in the peritoneum, less frequently in the pleura, and only very rarely in the pericardium. They sometimes occur simultaneously in both peritoneum and pleura. Chylothorax is undoubtedly a rare condition, and in a recent paper Dr. Mackenzie Wallis and Dr. Schölberg[2] were only able to collect sixty cases which have been recorded since the year 1860.

Effusions of this nature may be divided into two groups: (1) *True chylothorax*, (2) *Pseudo-chylothorax*, in both of which the naked-eye appearance of the fluid may be very similar. In the first variety, *the true chylous variety*, the fluid has become milky owing to the fact that as a result of injury or pressure upon the thoracic duct, a rupture of its walls has occurred, and chyle has found its way into the pleural cavity. The milky colour, in fact, is due to the presence of free fat. In the *pseudo-chylous form*, on the other hand, as Dr. Schölberg and Mr. Mackenzie Wallis have shown, the milky appearance results from the presence in the pleural fluid of a special compound or "complex," a lecithin-globulin or, as in the case recorded in this chapter, a cholesterin-globulin compound, of which we shall speak more fully later. The physical and chemical characters of the fluids in these two groups are somewhat different, and, following the investigations by Dr. Schölberg and Dr. Mackenzie Wallis, may be briefly given as follows:

The True Chylous Variety.

1. The fluid tends to accumulate rapidly, and in consequence large quantities are removed by paracentesis.

2. In colour it is generally yellowish-white. Emulsification is not perfect.

3. *The degree of opalescence is more or less constant* at successive tappings.

4. *The fluid possesses an odour* corresponding to the odour of the food digested.

5. Putrefaction occurs on standing.

6. A creamy layer generally forms on standing, owing to the amount of fat present.

7. The specific gravity generally exceeds 1·012.

8. The freezing point is depressed about –0·51° C., the figure thus approximating that for chyle.

9. No change occurs on filtering through filter paper. On centrifugalisation the fluid creams and may partially clear. On filtering through a Pasteur candle the milky colour remains.

10. Microscopically, *many fine fat globules,* staining with osmic acid and Sudan III. are seen, but very few cellular elements.

11. *On shaking the fluid with ether and a little potash the fat is dissolved* and *the fluid clears.*

12. The total solids vary considerably, but are usually greater than 4 per cent.

13. The total protein-content generally exceeds 3 grm. per cent., and of this amount serum-albumen forms the largest fraction, globulin occurring only in traces.

The Pseudo-chylous Variety.

1. The fluid collects more slowly, the volume of the fluid varying with the exciting pathological condition.

2. In colour it is pure milky-white. The solution forms an almost perfect emulsion.

3. *The opacity often increases or diminishes at successive tappings;* and may entirely disappear.

4. *The fluid is odourless.*

5. *It long resists putrefaction,* probably owing to the presence of lecithin.

6. A cream may or may not form, and this does not affect the opalescence. A sediment frequently settles out.

7. The specific gravity is generally less than 1·012.

8. The depression of the freezing point ranges from –0·56° to –0·61° C., thus corresponding to the figures for blood-serum.

9. No change occurs on filtering through paper, nor on centrifugalisation; but on filtering through a Pasteur candle the fluid clears.

10. Microscopically, the quantity of free fat is variable; *numerous fine, highly refractive granules* (lecithin-globulin complex) are seen, which *do not stain like fat.*

Cellular elements may be numerous and often contain fat; sometimes they are scanty.

11. *Shaking with ether and potash* may dissolve some fat, but the *opalescence remains.*

12. The total solids rarely exceed 2 per cent.

13. The protein-content varies between 1 and 3 per cent., the serum-globulin occurring in appreciable quantities.

The True Chylous Variety.	*The Pseudo-chylous Variety.*
14. Mucinoid substances are absent.	14. Mucinoid substances are sometimes present.
15. *The fat-content is generally high,* varying from 0·4 to 4 per cent. The fat corresponds in all its properties to the fat contained in food.	15. *The fat-content is generally low* and may be present in traces only; in its melting point and chemical composition it proves to be pathological fat.
16. Of the lipines, cholesterin is invariably found, and *lecithin only occurs in traces.*	16. *The most characteristic lipine is lecithin;* cholesterin is occasionally present.
17. There is no evidence of the presence of a lecithin-globulin complex.	17. *The lecithin is mainly combined with the globulin, and the suspension of this complex is the cause of the milky appearance.* If this complex be removed by filtration through a Pasteur filter, or be precipitated by half-saturation of the fluid with ammonium sulphate, or as the result of the removal of the salts by dialysis, the opalescence at once vanishes.
18. The salts and organic substances present approximate the values found for chyle obtained from the thoracic duct.	18. The salts and organic substances correspond closely to those of lymph and serous fluids.

NOTE.—*The figures given for specific gravity, total solids, and total protein-content must be taken as relative only,* and may be found considerably higher if the pleura be the seat of much inflammatory change.

We thus see that in the true chylous variety the opalescence is due to the presence of chyle, and to the large amount of fat present, under the microscope many fine fat globules being seen, which stain with osmic acid and Sudan III., and often show Brownian movements. If the fat be removed by shaking with ether and potash, the opalescence vanishes. In the pseudo-chylous form, on the other hand, though fat may be present, the opalescence is not due to it, and does not disappear on shaking with ether and potash, but results from the suspension in the fluid of a combination of globulin with lecithin (or cholesterin), which appears under the microscope as numerous fine, highly refractive granules, which do not stain like fat. Dr. Schölberg and Dr. Mackenzie Wallis proved this by half saturating the milky fluid with ammonium sulphate, a precipitate being produced, which on investigation proved to be composed of globulin and lecithin. On filtering the precipitate a clear fluid resulted. A similar

result also followed the filtration of the milky fluid through a Pasteur filter, which kept back the lecithin-globulin complex, and yielded a clear filtrate. That the above complex is held in suspension by the inorganic salts present was proved by exposing the fluid to dialysis. As the salts were in this way removed the complex was precipitated and the fluid cleared.

Ætiology.—Of the two varieties of chylothorax, true and false, the pseudo-chylous form is probably the one most frequently met with.

The causes which produce chylothorax of the true chylous type may be reduced to two:

(*a*) *Increased pressure within the thoracic duct*, for the most part the result of obstruction due to malignant growth (more rarely filarial in nature), leading to backward flow of the chyle along the pulmonary and pleural lymphatics, whence access to the pleural cavity is easy.

(*b*) *External violence resulting in rupture of the thoracic duct* and presumably of the pleura.

Of these two causes the former is the more important.

The pseudo-chylous form would seem to occur most often in connection with *malignant disease* and *tuberculosis*, and it has been suggested that the lecithin, the presence of which in the fluid we have seen to be so important, is set free as the result of cell-destruction taking place during the course of the disease. The lecithin subsequently finds its way into the serous cavity, where it unites with serum-globulin to form the complex which produces the milky appearance.

A study of the literature shows that, taking all cases of chylothorax together, nearly half are found to be dependent upon new growth, tubercle and injury being the next most common causes.

Chylothorax occurs in either sex and at all ages. It is, however, somewhat more common in the male, partly perhaps as the result of the greater liability of males to injury.

As regards age, the youngest case recorded is that of a child, aged five months, the condition being the result of injury; the oldest patient was aged sixty-seven, the chylothorax in her case following upon new growth. Thirty-one cases out of forty-eight occurred between the ages of twenty and fifty.

Symptoms.—The symptoms of chylothorax are in no wise distinctive. The side gradually fills with fluid, and, if sufficient in amount, displacement of the heart follows. The temperature is generally not raised, and the patient complains of little or nothing beyond some shortness of breath on exertion. It is not indeed until an exploratory puncture is made that the peculiar character of the fluid is recognised. Its nature might be suspected, however, if a fluid of like character had already been withdrawn from the peritoneum.

Prognosis.—The prognosis in a case of chylothorax of whatever type is serious, the primary cause of the condition, whether growth, tuberculosis, or injury, being in itself of serious augury. The presence of the milky effusion does not materially add to the gravity of the condition, unless the resulting respiratory distress calls for repeated paracentesis, in which case the drain of fluid from the circulation may hasten the downward course of the original malady.

Recovery, however, in cases not malignant in nature, is by no means unknown. It has been met with in many cases produced by injury, and may occur in others secondary to disease. Thus in a case recorded by Mr. Penn Milton,[3] which occurred in a patient suffering from early phthisis, recovery ensued after a single aspiration, in which 300 ounces of milky fluid were removed from the right side of the chest. Again, in a boy, aged thirteen, suffering from Hodgkin's disease, whom one of us had the opportunity of seeing whilst under the care of Dr. Ormerod[4] at St. Bartholomew's Hospital, the chylothorax—in this case of the true chylous variety and on the right side—disappeared after three aspirations had been performed. In this and similar cases the recovery is probably due to the enlargement of the anastomosing lymph branches, which connect the thoracic with the right lymphatic duct.

Diagnosis.—The milky fluid withdrawn in cases of chylothorax has been mistaken for thin pus, and we have known resection advised in such a case before the mistake was realised. The distinction is readily made if attention be given to the following points. If the fluid be pus, (1) numerous cells will be seen under the microscope, the number corresponding to the opalescence. (2) Centrifugalisation will yield a deposit of cells at the bottom of the tube and a clear supernatant liquid. (3) Filtration through filter paper will

keep back the cells and yield a clear filtrate. In all these points the purulent fluid differs from the milky fluid of chylothorax.

We may perhaps mention here, as causing a possible error in diagnosis, that anomalous condition described by Dr. Samuel West,[5] in which the pleura contained several pints of cream-like fluid, the appearance of which was due to numerous bright refractive globules composed chiefly of calcium phosphate. At the autopsy, which was made by one of us, numerous larger irregular masses of similar structure were found, mostly free, but some attached to the pleura, giving the pleural cavity, especially in its basal portion, the appearance of a "chalk-pit."

Treatment.—In chylothorax, whether of the true or false variety, we should not be in too great a hurry to intervene. In the true chylous form, if the heart be much displaced and dyspnœa urgent, paracentesis must be performed; otherwise it is best to wait, and to trust to the establishment of the collateral circulation. As we have seen, this may occur even after one or two tappings. In cases of the pseudo-chylous variety the effusion should be treated on ordinary lines as though it were a simple serous effusion, the milky character altering neither the outlook nor the treatment of the condition. Aspiration should therefore be performed from time to time as occasion requires.

Notes of a Case of Pseudo-chylothorax occurring in a Patient suffering from Hodgkin's Disease.[6]

We may perhaps focus what we have said by briefly describing the following case of pseudo-chylothorax, the patient having been under our care in the Brompton Hospital:

A. H., aged thirty-five, railway guard, was admitted into the Brompton Hospital on September 12, 1913, complaining of weakness and pains in his chest. He had suffered in 1901 from left-sided pneumonia and pleurisy, but from this he completely recovered and remained well until March, 1913, when he began to complain of debility. He continued his work until July, when, owing to increasing weakness and pain in the left side of the chest, he was obliged to relinquish it. About this time also he began to complain of cough and expectoration and to suffer from shortness of breath and night sweats. There was some little loss of weight. On September 12 he was admitted into the hospital.

Condition on Admission.—The patient looked ill. His temperature was swinging, rising at night to between 100° and 101° F. Pulse, 120. Respiration, 32.

On examining the chest the signs of a large left-sided pleural effusion were observed. The left side was dull from apex to base, with very weak breath-sounds and diminished vocal vibrations; the heart was displaced about a finger's breadth to the right of the sternum. The note at the right apex was slightly impaired, but no added sounds were audible. The abdomen contained some little free fluid. The spleen was markedly enlarged, the organ reaching down to the umbilicus and presenting a very definite edge. There was no enlargement of the external lymph glands. The bowels were somewhat loose. The urine and other organs were natural.

On September 13, the day following his admission, the left side was explored and a syringe full of milky fluid withdrawn. This presented a pale yellowish-white colour, and suggested at first sight pus, but proved on analysis (see p. 174) to be a pseudo-chyothorax. On September 19 aspiration was performed, and 49 ounces of a similiar fluid were evacuated. The note now became resonant in front to below the nipple, but the breath-sounds remained weak.

The opalescent fluid withdrawn was sterile, and gave the following differential count: 80 per cent. small lymphocytes, 10 per cent. large lymphocytes, and 10 per cent. polymorphonuclear cells; and as about this time tubercle bacilli were found in the sputum, the case was considered to be one of pulmonary tuberculosis, with tuberculous pleurisy, in which the fluid presented the pseudo-chylous character. The spleen was also regarded as tuberculous in nature, studded in all probability with caseous masses, although the rarity of such a condition in the adult was fully recognised.

After the paracentesis the temperature continued hectic and the side rapidly refilled, the heart becoming again displaced, and the left side measuring $\frac{3}{8}$ inch more than the right.

On September 26 paracentesis was performed a second time, and three pints of milky fluid withdrawn. The cytological count was very similar to that found on September 19; no tubercle bacilli could be discovered in the fluid. The blood-count on October 2 showed 3,700,000 red cells; hæmoglobin, 80 per cent.; leucocytes, 4,000. The differential count showed some diminution in the lymphocytes, with corresponding increase in the polymorphonuclear cells, but was otherwise normal.

On October 7 paracentesis was again performed, and three pints of opalescent fluid withdrawn. On cultivation this proved to be sterile, and no tubercle bacilli could be found in it. Cytological examination showed it to contain: small mononuclears, 81 per cent.; large mononuclears, 10 per cent.; polymorphonuclears, 9 per cent.

The temperature still continued to swing, reaching 100° F. at night, but the patient suffered little. Towards the end of the month dyspnœa returned (respirations, 32; pulse, 128), and as the heart was displaced paracentesis was performed on October 29

for the fourth time, three pints of the opalescent fluid being removed. From this date the patient gradually became weaker and was troubled with diarrhœa. The chest filled again, and there was some dyspnœa, but no real distress. At the end of November he became worse, with delirium and incontinence of urine and fæces, and died peacefully at 7 p.m. on November 30.

Post-Mortem Examination.—The autopsy, made by Dr. R. A. Young, showed that the diagnosis of tuberculosis made during life, based largely on the reported finding of tubercle bacilli in the sputum, was incorrect. No evidence of tuberculosis could be discovered in lungs, pleuræ, or spleen, but the patient was found to be the subject of a diffuse and extensive growth, thought at first to be sarcomatous, but which microscopical examination proved to be lymphadenomatous in nature. The following are the more detailed findings:

Body.—Much emaciated.

Chest.—On opening the chest the right pleural cavity was found to contain 8 ounces of clear serous fluid—the pleura itself was natural. The left pleural cavity contained about two pints of slightly opalescent fluid, much less milky than on the occasion of the last paracentesis, a month before death. Dense adhesions were present at the apex, and one band of adhesions was present over the lower lobe, but the pleura was elsewhere shiny and appeared natural.

The Lungs.—The left lung was markedly collapsed, and on section was slaty blue and airless. The right lung showed a considerable degree of emphysema, and was very œdematous throughout; a few adhesions were present at the apex. No tuberculous foci were found in either lung. The larynx, trachea and bronchi were natural. The bronchial and anterior mediastinal glands were markedly enlarged, and showed secondary deposits of growth, but no naked-eye evidence of tubercle. The pericardium and heart were natural.

On removing the heart and lungs a mass of growth, of firm consistency, was seen to extend over the four lower thoracic vertebræ, and to form a sheath over the lower and contiguous portion of the thoracic aorta. It also extended for some little distance under the adjacent portion of the left pleura. The thoracic duct was not discovered, being embedded in the growth around the aorta and in the dense adhesions at the apex of the left lung.

Abdomen.—The peritoneal cavity contained about 100 ounces of blood-stained serous fluid. On removing the contents a large mass of growth was found, nearly 2 inches in thickness, surrounding the whole of the abdominal aorta and the common iliac arteries. The growth extended widely in all directions behind the peritoneum and infiltrated the bodies of the second and third lumbar vertebræ, which presented in parts a rough worm-eaten appearance. The left suprarenal gland was embedded in the growth and infiltrated by it.

The *liver* was enlarged and contained numerous soft vascular growths. A gland, enlarged by growth, lay upon the gall-bladder, and others in a similar condition were seen along the lesser curvature of the stomach. Those along the upper margin of the pancreas were

much enlarged and occupied by growth, but the pancreas itself was free.

The *spleen* was greatly enlarged, weighing 22 ounces. Its surface was irregular from the presence of numerous growths within its surface. On section it was found to be riddled with growths, which were dark in colour, probably from hæmorrhage into their substance.

The stomach, intestines, bladder, prostate and testicles were normal; the kidneys were enlarged and slightly fatty. The receptaculum chyli could not be found, all the structures in its neighbourhood being densely infiltrated with growth.

The naked-eye appearances, thus described, suggested that the disease was a retroperitoneal sarcoma with numerous secondary growths. The following report, however, by Dr. Young upon the microscopical character of the sections showed that in fact it was a case of lymphadenoma :

Report upon the Microscopical Characters of the Growth by Dr. R. A. Young, M.D., F.R.C.P.

"*A Portion of the Main Retroperitoneal Mass of Growth.*—The section presented the characteristic appearances of true Hodgkin's disease or lympho-granuloma; the reticulum was abundant, and there were numerous typical giant cells. Under the one-twelfth power some of the cells in the reticulum showed eosinophile granulation. There was very little degenerative change, but some hæmorrhage was observed in parts of the section.

Sections of mediastinal and bronchial glands, and of the growths in the liver, spleen, and left suprarenal showed a similar miscroscopical character."

Report by Dr. R. L. Mackenzie Wallis upon the Milky Fluid withdrawn from the Pleural Cavity.

"The fluid was milky in appearance, presenting a pale yellowish-white colour. It was odourless, and presented a specific gravity of 1021. Reaction alkaline.

It did not cream on standing. The opalescence was not removed by filtration or centrifugalisation, but was removed by passage through a Pasteur filter. Chemically its composition was as follows .

Total solids	4·560 per cent.	
Inorganic ash	0·821 ,,	
Serum albumen	2·020 ,,	Total protein = 3·57 per cent.
Serum globulin	1·55 ,,	
Total nitrogen	0·58 ,,	
Fat and lecithin	0·06 ,,	
Cholesterol	0·052 ,,	
Sugar	absent.	
Nucleo-proteid	absent.	
Mucin	a trace only.	
Sodium chloride	0·43 per cent.	
Ratio of albumen to globulin, nearly	2 : 1.	

Conclusion.—The fluid in its physical properties behaves exactly like a pseudo-chylous fluid—*i.e.*, it does not cream, the opalescence is unaffected by filtration or centrifugalisation, but is removed by passage through a Pasteur filter. The opalescence is apparently due to the occurrence of globulin united to cholesterol and lecithin, the former being in greater quantity. In its chemical composition and physical properties the fluid behaves exactly like a pseudo-chylous fluid, but differs from those previously described in the preponderance of cholesterol over lecithin—the figure for the lecithin being too small to warrant inclusion as a definite entity.

The fluid would appear, therefore, to be a true pseudo-chylous pleural fluid, and not derived from rupture or obstruction of the thoracic duct."

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CHAPTER XII

BRONCHITIS—BRONCHIAL CATARRH

ACUTE bronchitis consists in an active inflammation of the mucous membrane of some portion of the bronchial tract.

Ætiology.—The disease is especially prevalent in northern latitudes, in exposed and elevated situations, and in districts where moisture of atmosphere as well as a low temperature and cold winds prevail. In our British climate these conditions are but too well fulfilled, and bronchitis is endemic amongst us.

January, our coldest and dampest month,* is that in which it is most prevalent; but from the variable temperature and cold winds which characterise our spring, bronchitis is then also of common occurrence. In our autumn it is less prevalent, although persons with a tendency to recurrent bronchitis are often attacked each year as the colder weather comes round.

The incidence of bronchitis is influenced by *age, sex, occupation,* and *conditions of life,* chiefly in so far as these circumstances favour exposure to the known exciting causes of the disease or diminish the resisting power of the individual.

During the first dentition some children have repeated attacks of bronchial catarrh coincident with the eruption of each tooth, just as other children suffer from catarrh of the nasal or intestinal tract, and from the time of dentition onwards throughout childhood bronchitis is common. This is to be accounted for partly on grounds of lessened power of resistance, partly in consequence of dentition itself, and in part also from the frequently impaired nasal development

* The statistics of Mr. Campbell Bayard[1] show that at nearly all the English stations the mean temperature, as also the mean minimum temperature, reaches its lowest in January, and the mean relative humidity its maximum.

and obstructed nasal passages of children, which necessitate oral breathing. Adenoid growths in the posterior nares are the most common cause of such obstruction in both sexes, and are especially prevalent in the Jewish race. The abnormal method of breathing thus entailed not infrequently remains a bad habit through life, and is a fertile source of chest troubles by the direct manner in which the air is permitted to enter the bronchi and lungs, unwarmed and unfiltered by its passage through the nares.

Old people have but feeble powers of resistance, and bronchitis is very prevalent and fatal amongst them. The male sex is more exposed to the causes which lead to bronchitis than the female, and suffers more accordingly.

Cachexia of various kinds, gout, syphilis, phthisis, alcoholism, or Bright's disease, must also be ranked amongst the predisposing causes of the disease, one or other of these maladies being often at the root of the more protracted and recurrent cases. Certain forms of heart disease, such as mitral regurgitation and, in a still greater degree, mitral stenosis, also predispose to bronchitis by obstructing the return of blood from the lungs, thus causing mechanical congestion of these organs and of the smaller bronchi.

Heredity has a certain influence in the causation of the disease. Recurrent bronchial catarrh, associated with asthmatic symptoms, and with more or less emphysema, is well known to be an hereditary affection, and may occur very early in life. The winter bronchitis to which many individuals become subject at certain periods of life also runs markedly in families, and is, no doubt, really a phenomenon of premature senility. But, with these exceptions, it cannot be said that bronchitis is hereditary, and our own observations would lead us to the opinion that, when the predisposition is apparently seen running through the children of certain families, it is often connected rather with enlarged tonsils or imperfect development and adenoid affections of the nasal passages, which necessitate oral breathing, than with any inherited delicacy of the bronchial membrane.

Of all the exciting causes of bronchitis, depression of temperature is the most important. The attack commonly supervenes upon exposure to sudden changes of temperature or to cold, wet winds, especially in depressed conditions of the

system, when the patient has been overheated by exertion or exhausted by mental fatigue or shock. A draught of cold air, affecting a very limited portion of the body, or the chilling of the feet from sitting in wet boots may be sufficient to produce it. Bronchial attacks frequently begin with "colds in the head," and many people have a morbid state of the naso-pharyngeal membrane, which tends to the production of stagnant and unhealthy mucus, harbouring the germs of catarrhal and influenzal diseases. This is an important point to remember in the treatment, or rather the prevention, of recurrent bronchial catarrh.

Another factor of great importance in the causation of the disease is the inhalation of irritating particles, and certain occupations which are very dusty prove harmful in this respect. These trades have been divided by Hirt[2] into five classes, according as the dust is (1) metallic (as with file-cutters and knife-grinders); (2) mineral (as with potters, masons, and cement-workers); (3) vegetable (as with coal-miners, tea-packers, fret-cutters, and flax-dressers); (4) animal (as with wool-carders and furriers); and (5) mixed (as with street-sweepers)—an interesting classification, in that it indicates how various are the kinds of dust which may be at fault and how numerous are the occupations affected. In a later chapter (Chapter XV.) we shall describe a case of dust bronchitis and asthma produced by the inhalation of rosewood dust. It is instructive to note, however, that of those who are exposed to irritating mechanical influences of this kind many escape unharmed; and Hirt has further observed that among those at first attacked the inhalation, if continued, may after a time fail to excite catarrh, the workmen becoming, as it were, acclimatised to their conditions. On the other hand, in noting the history of patients who have suffered from irritative bronchitis, we have found instances in which the fathers of the patients had passed their lives at the same occupation without complaint, the second or third generation thus seeming in some cases to become more vulnerable to the given influences.

But the evil effect of dust is not limited to certain occupations. Many a fresh catarrh in cases of confirmed pulmonary disease may be traced to dusty winds or to the irritating fogs, which so frequently prevail in London and its neigh-

bourhood, and which prove so deadly a scourge to elderly bronchitic subjects.

Bronchitis, again, is closely connected with many of the specific fevers. Thus, measles, whooping-cough, typhoid fever, and smallpox are attended by it usually in the early periods of the disease. Bronchial catarrh, sometimes of a very obstinate character, and yielding only to specific treatment, is a not infrequent concomitant of the eruptive period of syphilis; and in influenza acute bronchitis often forms an important element in the disease.

Blood mingled with septic matter inhaled into the bronchial tubes during an attack of hæmoptysis or during tracheotomy may set up bronchitis, and a virulent form, attended with profuse expectoration, is occasioned by the passage over the bronchial surface of acrid septic matters from fœtid pulmonary or pleural cavities. Bronchitis and broncho-pneumonia are also well known and sometimes fatal complications of surgical operations, the irritating effects of certain anæsthetic vapours, such as ether, upon the bronchial mucous membrane, and the aspiration of saliva into the air passages during the administration of the anæsthetic, especially when this is accompanied by much struggling and cyanosis, being probably the determining factors.[3]

Bacteriology.—In the preceding paragraphs we have enumerated the more important factors concerned in the production of bronchitis. It must be clearly understood, however, that for the most part these are but predisposing causes which, by their weakening influence, favour the growth of micro-organisms which play a preponderating rôle in the development of the disease.

Of the organisms which may be at fault, the pneumococcus and the streptococcus are the most important; but Pfeiffer's bacillus of influenza and the micrococcus catarrhalis are not infrequently present, and no doubt take their share in the ætiology of the disease. Occasionally other organisms are found in the bronchial secretion, such as the staphylococcus aureus and albus, the bacillus pyocyaneus, or the diphtheria bacillus, the latter even in the absence of any clinical evidence of diphtheria. In certain cases one or other of the micro-organisms mentioned may be found in pure culture in the bronchial exudation and must then be held responsible for

the attack; but in the majority of cases this is not so, and the bronchitis—at all events, in its later stages—results from a mixed infection by two or more organisms, among which the pneumococcus, streptococcus, Pfeiffer's bacillus, and micrococcus catarrhalis figure most prominently.

In tropical climates, as shown by Castellani[4] and others, bronchitis is sometimes produced by a spirochæte (*Broncho-spirochætosis*), the organism being found in large numbers in the sputum. It would appear probable also that in these climates bronchial catarrh is not uncommonly due to the presence in the air passages of hyphomycetes and other fungi (*Broncho-mycosis*).[5] The so-called "tea-factory cough," common in Ceylon, is probably an affection of this nature.

Morbid Anatomy.—The characteristic appearances of acute catarrh are but rarely to be observed post-mortem, but they may sometimes be seen in perfection in the trachea during life by means of the laryngoscope.

1. In the first stage there is hyperæmia of the mucous membrane, with œdema of the basement layer. The affected membrane is minutely injected, swollen, and unduly lacerable, and presents in severe cases, especially in young children, minute hæmorrhages. Upon hyperæmic swelling of the mucous follicles depends in part the temporary check to secretion, which characterises the early stages of bronchitis, the ducts of the follicles being also to some extent occluded by the swollen condition of the mucous membrane which they traverse.

2. The mucous flow is soon increased, however, and is mingled with sanious exudation from the vessels, sheddings of columnar epithelium, cubical or small round cells derived from multiplication of the deeper cells of the epithelial layer, and with pus cells. Thus is constituted the muco-purulent secretion of bronchitis, thin and glairy at first, thicker and more opaque after a few days. To the naked eye the appearance of the mucous membrane is now more slaty in hue, covered with secretion, sanious or thick, according to the stages referred to.

3. The tissues of the bronchial tubes beneath the basement membrane do not remain passive whilst these active processes are going on in the layers above them. As demonstrated by the late Professor Hamilton,[6] the connective-tissue cells

multiply, and the endothelium of the lymphatic spaces of the inner and outer fibrous layer proliferates. The products of such proliferation do not, however, find their way to the surface, being unable to penetrate the basement membrane, but collect in the interstices of the cellular tissue, and fill up the lymph paths along which they slowly course towards the bronchial glands. By these processes thickening of the bronchial membrane is effected, together with some induration of lung texture spreading from the bronchial sheaths.

Clinical Varieties of Bronchitis.

Like many other diseases, bronchitis may present itself in an acute or in a chronic form; and these, again, may appear under various disguises. The following classification embraces the chief varieties met with in practice:

1. Acute Bronchitis—(*a*) *Acute tracheo-bronchitis* (affecting the larger and medium-sized tubes; the common form of the disease in the adult).

(*b*) *Capillary bronchitis, or suffocative catarrh* (occurring mostly in children, and affecting chiefly the smaller tubes).

(*c*) *Acute asthenic bronchitis* (occurring at the extremes of life, but especially in the aged. In it the toxic symptoms are marked).

(*d*) *Purulent bronchitis*—an acute variety of bronchitis, occurring sometimes in epidemics, and marked by a copious purulent expectoration, in which Pfeiffer's bacillus of influenza is often found. It is met with, however, also in a chronic form.

2. Chronic Bronchitis—*Chronic muco-purulent catarrh.*

3. Peculiar Forms of Bronchitis (which may be either acute or chronic).—

(*a*) *Pituitous catarrh, or bronchorrhœa serosa.*

(*b*) *Bronchitis sicca—dry catarrh—catarrhe sec.*

(*c*) *Plastic bronchitis.*

Acute Tracheo-Bronchitis.

The symptoms which usher in an acute catarrh of the large bronchial tubes—the form in which bronchitis usually manifests itself in the adult—are commonly those of an ordinary cold in the head. Chills of a creeping character, never

amounting to a rigor, occur from time to time on the first day, and are attended with a feeling of malaise, a somewhat hurried pulse, slight soreness of the throat, sneezing, and coryza. The temperature is raised a degree or so above the normal, but, although there is thirst, aching, perhaps pains in the limbs, and a considerable sense of feverishness, the febrile phenomena are really very slight in adults, though more decided when the disease occurs in young children. After some twenty-four or forty-eight hours, the patient complains of a soreness, or, as it is often more accurately described, a rawness behind the upper sternum, accompanied by a sense of constriction or oppression in this region. The cough is frequent and dry, and is attended with more or less pain of a rending character. The voice is deepened, and sometimes husky or suppressed, and the breathing is perceptibly quickened. On the second or third day secretion takes place, and with the expectoration of a thin aërated mucus the patient soon experiences a marked sense of relief. In fact, the pyrexial stage has already passed, the pulse is quiet, the cough loose, and expectoration easy, the mucus expelled becoming more opaque and semipurulent. Gradually the secretion subsides and the cough lessens, until in a week or ten days it disappears, except that in the morning, on first awaking, the patient still feels some oppression in the chest, which is not relieved until he has brought up some purulent mucus.

As regards physical signs, we may hear a few dry, sonorous rhonchi vibrating through the chest, obviously produced in the larger tubes; but in some cases, when these tubes alone are affected, there may be a total absence of all physical signs. When the secretion is more established, the rhonchi, which are symmetrically distributed over both lungs, become looser, modified or, for the moment, removed by cough, and accompanied, especially over the base of the lungs, by scattered, muffled, bubbling râles. The percussion note over the chest is unaltered.

Capillary Bronchitis, or Suffocative Catarrh.

This form of the disease may be met with at all ages, but is most frequently seen in children. In them the power of coughing is at a minimum, and the septic secretion of the

larger bronchi, in which the disease begins, finds its way rapidly through deep inspiratory efforts or by gravitation into the finer tubes. These quickly become blocked, and the characteristic signs of the disease, merging insensibly into those of broncho-pneumonia, become developed. Thus dyspnœa soon becomes marked, the nares expand with each inspiration, the lips are cyanosed or even livid, and the face assumes an anxious and distressed expression. The temperature usually reaches 101° or 102°, although in feeble or old people there may be considerable systemic shock, with general prostration and reduced temperature. The pulse-rate is increased, but not in proportion to the respiration. The cough is frequent, and is very soon accompanied by the expectoration of a viscid adhesive mucus difficult to dislodge. The digestive functions are impaired, the tongue furred, the bowels confined, and the urine often loaded with lithates.

On inspecting the chest, the thoracic movements are observed to be increased, since it is mainly with the comparatively unaffected front and upper portions of the lungs that the patient breathes. The resonance on percussion is everywhere unimpaired, and it may be even increased. Fine bubbling râles are audible over both posterior bases, and to a much less extent, or not at all, over the upper and anterior portions of the chest, where, however, sibilant and sonorous sounds prevail. This distribution of the râles in bronchitis is mainly a question of gravitation, and, as the late Dr. Walshe pointed out, even in bronchitis of mild type, and not involving to any extent the capillary tubes, we may still hear fine râles at the posterior bases from gravitation of the secretion to the smaller tubes. After a few days, if the patient have sufficient strength, the signs of the disease gradually lessen, and convalescence in due time becomes established; but the bronchi long remain weakened and liable to a fresh attack, unless carefully shielded from exposure. In severe cases, after struggling for some days, the signs of cyanosis and asphyxia increase, the patient passes into a drowsy condition, and death soon follows.

The diagnosis of bronchitis of this degree depends upon the symmetrical distribution of fine bubbling râles not associated with any percussion dulness or bronchial quality of breath-sound, and with but a moderate rise of temperature.

The diseases which may be confounded with it are pulmonary œdema, miliary tuberculosis of the lungs, and acute phthisis.

If œdema be limited to the lungs it must be dependent upon cardiac defect, the history and signs of which render the case clear.

Miliary tuberculosis, on the contrary, may be easily mistaken for bronchitis, since the physical signs are almost identical. Nevertheless, careful examination will put us on our guard. Thus, it is not uncommon in tuberculosis to find that, though the lungs are stuffed with miliary tubercles, there may be comparatively few moist sounds, and these often quite as marked at the apex as at the base. If, therefore, in a given case we find excessive dyspnœa, cyanosis, and great prostration, apparently out of all proportion to the physical signs, the suspicion of miliary tuberculosis of the lungs should at once suggest itself. The temperature is not distinctive, since in tuberculous cases it is by no means necessarily high. Evidence of previous pulmonary disease at one apex would cause the diagnosis to lean strongly towards tuberculosis.

In children especially, acute phthisis of the caseous broncho-pneumonic type may lead to difficulty in diagnosis. This variety of the disease not infrequently follows whooping-cough or measles, and, from the diffuse signs of bronchitis to which it gives rise, may for a time be confounded with simple catarrh. Later, more or less pneumonic crepitation, patchy, tubular breathing and larger clicks, are superadded to the simple râles, and the signs become distinctly more advanced at some one portion of the chest, whether base or apex. But here again the diagnosis between a simple bronchitis passing on into broncho-pneumonia and the tuberculous variety of the disease is by no means easy. The history of the onset after measles or whooping-cough, the longer duration of the disease, and its steadily downward course, should suggest its tuberculous nature. A bacteriological examination of any sputum that can be obtained or of the fæces should by no means be neglected.

Acute Asthenic Bronchitis.

This is a malady commonly met with at both extremes of life, and in the aged especially it is one of the most fatal of

diseases. It is marked chiefly by the rapidity of its course and the severity of its toxic symptoms. The following fairly characteristic example may be related in illustration of the salient features of the disease:

Mrs. M——, a widow, aged seventy-six, of thin, spare build, and of previous good health, was dining out with friends on the evening of December 27, feeling in her usual good health and spirits. She had never before suffered from any chest illness, but it was believed that on her way home she became chilled. Mrs. M——, however, appeared well the next morning, but towards the latter part of the day (28th) felt drowsy and somewhat chilly. She became more obviously ill in the course of the evening, the breathing being quickened and oppressed, and towards midnight her symptoms became so aggravated that the doctor was sent for. She was now found to be in a state of profound collapse, with small, feeble pulse, cold extremities, low temperature, and sweating surface. The respirations were quick and shallow, the countenance anxious, and the mucous membranes somewhat cyanosed. No morbid sounds save a few wheezing rhonchi were to be heard over the chest.

A free recourse to brandy and other stimulating remedies rallied the patient so notably that the friends became hopeful, and the doctor plied his remedies and planned out food, physic, and stimulants with a cheery exactness. By the following evening the chest signs had become more marked, short inspirations being followed by prolonged wheezing expirations, the pulse keeping steady at about 90 beats a minute. The cough and expectoration were now troublesome and difficult, preventing, as the night advanced, more than brief snatches of sleep. The body temperature rose to about 100°, and it was observed that the patient wandered in slight delirium, from which, however, she could readily rouse herself. Fine bubbling râles were now, at the close of the second or third day, audible over the chest, most abundantly and most definitely at the posterior bases, where but very little air could be heard to penetrate. The respiratory movements were peculiar, at each inspiration the chest being lifted quickly by the action of the auxiliary muscles, whilst the bases of the thorax receded. A long wheezing expiration followed each inspiration, apparently produced by the downward recoil of the chest upon the diaphragm, which had been pushed upwards to meet it by the contraction of the abdominal muscles.

Tracheal rattles soon became developed, removable at first by cough, but soon to return. The pulse quickened, the patient became more exhausted, with muttering delirium or incoherence, and death closed the scene at about the seventieth hour.

Such is a sketch of the phenomena presented by a case of this fatal malady, the characteristic features being: (1) the almost fatal collapse at about the twelfth hour from the first

shock of the disease; (2) the rallying of the patient, and the oncoming of the signs of general bronchitis, attended with slight febrile reaction; (3) at about the third day the appearance of laboured, ineffectual breathing, with signs of filling of the lower bronchi, indicative, no doubt, of exhaustion of the nerve centres. The forcible respiratory efforts, far beyond the strength of the patient, which appear at this last and fatal stage, and continue to the end, are very noticeable, and it is rare indeed for recovery to ensue in any case in which this character of breathing has once been observed.

But such grave forms of the disease are not limited to the aged, for young children not infrequently succumb to the first shock of bronchitis, being overwhelmed before their illness seriously attracts the attention of their parents. A considerable proportion of the infants brought to our hospitals dead, or in a dying condition from sudden illness, are found post-mortem to present no other lesions beyond the signs, very slightly marked, of early bronchitis. A few petechial spots in the mediastinum, pericardium, or pleura, may testify to the brief struggle of the little patients.

Purulent Bronchitis.

Attention has recently been drawn to a form of acute bronchitis, which has occurred in epidemic form among our troops both in France[7] and in England,[8] and which is characterised by the expectoration of abundant nummular sputa composed almost entirely of pus. The amount may reach as much as fifteen ounces in the twenty-four hours.

These cases, first described by Drs. Hammond, Rolland, and Shore,[7] are sometimes very acute, and for a time suggest pneumonia; but the sputum, never really rusty, soon acquires its characteristic appearance. Tachycardia, dyspnœa, and cyanosis, often of a "heliotrope" tint, are prominent symptoms, and the patient not uncommonly dies in about a week from cardiac failure, the smaller bronchi and bronchioles being found after death to be filled with purulent exudation. In other cases, less acute, the malady may last some weeks, the temperature varying from 101° to 103°, and here again cyanosis and respiratory distress are marked features. In the early stages moist sounds are heard scattered over the lungs, to be succeeded later by signs of broncho-pneumonia.

In such cases the patient wastes and often sweats, and the disease may be mistaken for acute tuberculosis. The mortality in the epidemics which we have been describing has been severe, but of the less acute cases many recover.

Bacteriologically the cases are of interest, for in the majority the Pfeiffer's bacillus of influenza has been found in the sputum, associated in many instances with the pneumococcus. In an epidemic occurring among a draft of troops from New Zealand,[9] in which the purulent bronchitis occurred as a complication of measles and rubella, Pfeiffer's bacillus and the streptococcus were the organisms most often present. In view, however, of the evidence recently brought forward by Sir John Rose Bradford and Drs. Bashford and Wilson,[10] suggesting that a filter-passing virus may be the cause of influenza, the presence of the Pfeiffer's bacillus cannot be held as proof that these cases of purulent bronchitis are truly influenzal in nature.

It must not be supposed, moreover, that "purulent bronchitis" is met with only in epidemics, though these have naturally directed especial attention to its occurrence. We have met with more than one instance of subacute bronchitis in which the sputum has had this special character, though the amount expectorated has not reached the large quantity recently described, and in which the pneumococcus was present in pure culture. We know also of the case of a gentleman, aged between eighty and ninety, who during the few years preceding his death had several attacks of subacute bronchitis, each accompanied by the expectoration of characteristic purulent sputum, which continued also between the attacks. On two occasions this was found to contain a pure culture of Pfeiffer's bacillus. Lastly, as we shall see, copious purulent expectoration is sometimes associated with the later stages of a chronic muco-purulent catarrh, and to this condition the terms *purulent bronchitis* or *broncho-blennorrhœa* have long been applied (see p. 191).

Prognosis in Acute Bronchitis.—From what we have already said it will be understood that the outlook in acute bronchitis of whatever degree, when occurring in adults, is, generally speaking, favourable. Capillary bronchitis in very old and very young people, and still more so the acute asthenic and "purulent" forms, are, on the other hand, fre-

quently fatal. Nevertheless, there is, perhaps, no disease the mortality of which is more influenced by treatment.

Treatment.—Taking capillary bronchitis in the adult as our text in regard to treatment, our first thought should be to see that the room is adequately warmed. For this purpose a fire and steam-kettle are necessary, so as to raise the temperature of the room to about 62°, and immense relief is given to the patient by this means alone. Due care must be taken, however, to insure a proper and constant supply of fresh air, as well as to preserve a uniform temperature. The use of the steam-kettle is not only to moisten the air of the room—itself a point of no small importance when the cough is irritable and the expectoration viscid—but because in most bedrooms it is the only possible means of raising the temperature and maintaining it during cold weather at the desired height.

A large mustard or mustard and linseed poultice should be applied to the front of the chest or to the back, and followed up by hot linseed applications or cotton-wool to the chest. In children a jacket poultice is often very useful, but one must not forget that, both in young or weakly children and in old people, a linseed poultice wrapped round the chest may be a very serious impediment to free thoracic movements, and in such cases it is often more judicious to have recourse to cotton-wool covered with oil-silk and an occasional mustard or mustard and linseed poultice to keep the blood determined to the surface. Poultices are indeed valuable in the hands of the careful and skilled attendant; otherwise, hot cotton-wool applications, with the occasional help of a stimulating poultice, are much to be preferred. Pine or thermogen wool are more stimulating than ordinary cotton-wool, and are to be recommended especially for old people. Where the constant attention of a skilled nurse is not possible, antiphlogistine applications are especially useful, not requiring frequent renewal. For convenience, and to save fatigue in the use of poultices, the nightdress should be cut down the middle, and brought together with tapes or safety-pins.

As regards drugs, a saline mixture with ipecacuanha is the best with which to begin. In strong adults antimony wine is very useful, especially in the early dry stage of the catarrh, and should be given in small doses at frequent intervals.

Apomorphine, in $\frac{1}{24}$ to $\frac{1}{12}$ grain doses, is often, and especially in cases where there is some degree of bronchial spasm, a valuable substitute for ipecacuanha or antimony in the prescription. In old people, on the other hand, carbonate of ammonia is generally required. The special danger in infants arises from the possible occurrence of pulmonary collapse and broncho-pneumonia. These are due to the inability of children to expectorate, and the consequent plugging of the bronchi with secretions, which are drawn into the corresponding alveoli during vain inspiratory efforts. The timely administration of ipecacuanha emetics, if the secretion be abundant, will in some cases avert this danger. Friction with stimulating liniments, such as the ammonia or acetic turpentine liniments of the Pharmacopœia, further diluted, if necessary, with an equal quantity of olive oil, is of great service in young children after the first stage has passed.

In old people danger arises principally from exhaustion, with atony of the bronchial tubes. To avert these dangers we must from the first support the patient by the frequent administration of nutritious liquids and by the timely employment of stimulants in carefully regulated doses. Of all alcoholic stimulants, brandy is certainly the best for this purpose, though we have at times found whisky, with hot peppermint-water, an excellent stimulating expectorant. The administration of opiate remedies in bronchitis should, as a rule, be avoided, and absolutely so in cases in which lividity of lips shows already defective aëration of blood. For young children and old people opium should never be used in bronchitis. As a sedative at night, bromide of ammonium, with aromatic spirits of ammonia, is one of the best we can choose. Chloral is not very suitable in acute cases, but a small dose combined with bromide of ammonium will suffice to give rest without risk of unduly depressing the heart's action. When there is much lividity, with restless, muttering delirium, oxygen inhalations may be used with much success, securing short, refreshing sleeps, and maintaining nerve and cardiac power. When the heart fails, and symptoms of overloading of the right ventricle present themselves, digitalis may be usefully given, stimulants must be persevered with, and dry-cupping may be tried with advantage. In certain cases in which there is marked venous plethora, with a weak and fail-

ing heart's action from an overburdened right ventricle, venesection is attended with manifest relief.

Next to avoiding a fatal issue, our efforts should be directed to prevent the case passing into the chronic stage, which is especially to be feared in those who have suffered from previous attacks. When the acute symptoms are past, some patients at once convalesce without any special treatment; in others the secretion continues abundant and purulent. In such cases the saline mixture must be given less frequently or changed for a more stimulating expectorant containing senega and ammonia; and a mixture containing some mineral acid, with calumba or quinine, may be taken twice a day. The turpentine acetic liniment is of great value in this stage, its usefulness being probably in part due to inhalation of the turpentine vapour. Meanwhile the bodily strength must be well maintained, and for this purpose cod-liver oil may be prescribed.

In cases in which there is a tendency to frequent catarrh the naso-pharynx should be looked to, and will often be found to be in a catarrhal condition and to lodge unhealthy muco-pus, which may be sometimes observed in the posterior naso-pharynx. We are in the habit of advising in such cases the use of a lotion consisting of chloride of sodium ʒi., salicylate of soda ʒi., or borate of sodium ʒi.ss., hazeline ʒiii., glycerine ʒiii., and rose-water to ℥viii.: one tablespoonful to a wineglass of warm water for a nasal douche: or a choice can be made from the various collunaria of the London hospitals.[11]

Chronic Bronchitis—Chronic Muco-Purulent Catarrh.

This is met with most frequently in elderly people, but it is by no means unknown in adult life, and occurs not very rarely even in children whose nutrition is markedly impaired. It may sometimes originate in an acute attack from which recovery has been incomplete, and may in this way be traced back to measles or whooping-cough, from which the patient suffered several years before. Many cases in the adult, however, cannot be traced to a primary acute attack, and the disease is then chronic from the commencement. This may be observed in patients who are otherwise healthy, but in some the disease is associated with a constitutional malady, such as gout or chronic nephritis. In not a few cases

alcoholism is the true factor in causation. In others, excessive smoking and inhaling or a dusty occupation are responsible for the continuance of the malady.

Cough and the expectoration of muco-purulent sputum, with possibly some dyspnœa on exertion, though the latter depends upon the resulting emphysema rather than upon the bronchitis itself, are the principal symptoms of which complaint is made. These are always more marked in winter than in summer, and in early cases they disappear entirely as the warmer weather comes round.

As regards physical signs, there are two factors responsible for them—first, the bronchial catarrh, with mucous secretion causing the breath-sounds to be modified by râles and rhonchi; and, secondly, more or less associated emphysema of the lungs, causing a tendency to extension of pulmonary resonance beyond its normal limits (see Emphysema).

At first the patient suffers but little inconvenience from his complaint. In the early morning, on first waking, he may be troubled with cough, but after the tubes have been freed from the secretion which has accumulated during sleep, he will remain practically undisturbed for the remainder of the day. But with the progress of the disease his hours of immunity become lessened, and this is especially the case whenever the weather is damp, cold, and foggy. The amount of cough will, however, vary to some extent, according to the character of the sputum. Whenever this is viscid and difficult to expectorate, the cough will become more trying; whenever it becomes more loose, the symptoms are alleviated.

The sputum in a typical case consists of mucus mixed with pus. Sometimes these are mingled more or less uniformly; in other cases the pus predominates, and is at times seen floating in separate coin-like masses in the mucus and saliva. Such "nummular sputum" does not necessarily indicate the existence of bronchial dilatation. At a later period of the disease the amount of sputum may be greatly increased, and the terms "purulent bronchitis," and "broncho-blennorrhœa,"* have been employed to indicate the condition (see p. 187). At other times the expectoration becomes offensive. In the

* Blenna, like phlegma, pituita, and mucus, was, as the late Dr. Gee[12a] reminded us, but one of the terms used haphazard by the ancient physicians to signify the sputum, whether transparent or opaque.

absence of bronchiectasis this symptom rarely lasts for more than a few days or a week, and then gradually passes away. It is of no serious import, and probably results from the sputum becoming temporarily more viscid than usual, leading to its retention within the bronchi sufficiently long for putrefactive changes to set in. The condition of the mouth and teeth as a possible source of the fœtor should be investigated.

Of the existence of a special form of *putrid or fœtid bronchitis*, in which, without any organic lesion, such as bronchiectasis or gangrene of the bronchial membrane, the secretion is continuously offensive, we do not feel convinced. A feculent odour is stated, however, to occur sometimes in cases of pulmonary streptotrichosis.

At first, as we have said, the patient's nights are but little disturbed by his complaint; but later, as the secretion becomes greater in amount, attacks of dyspnœa, which closely resemble the paroxysms of true asthma, break his rest. With sleep thus disturbed, and with the disease making gradual progress—now slowly, now more quickly—under the influence of intercurrent and more acute attacks, the patient's strength gradually ebbs, though it is surprising how long the combat is maintained. At length the fatal issue is ushered in by an exacerbation of the disease, or by gradual failure of the right side of the heart to withstand the extra strain thrown upon it by advancing emphysema.

Treatment.—In cases in which the cough is dry and there is difficulty in expectorating the phlegm, relief will often be given by a mixture containing vinum ipecacuanhæ, potassium bicarbonate, and potassium iodide, for one of which, in cases requiring stimulation, we may substitute ammonium carbonate. Chloride of ammonium is often also of great value. When, on the other hand, there is much muco-purulent expectoration, considerable advantage may be gained by the use of tar, of which good preparations are Guyot's Eau de Goudron (ʒi.) or Bell's Liquor Picis Aromaticus (♏ xx. to ʒi.), in plain or alkaline water, such as that of Vichy or Ems; or Jozeau's capsules may be preferred. In this stage, also, the resinous preparations, such as oil of turpentine and terebene, or the oil of sandal wood, are sometimes useful. For the paroxysms of asthma iodide of potassium and stramonium

will generally prove effective, as in simple asthma. We have not been greatly impressed with the value of vaccines in the treatment of chronic bronchitis.

But in many cases of chronic bronchitis it is best to leave the bronchial membrane alone, and to direct treatment towards improving digestion and appetite and the general nutrition. In this connection we must emphasise the value of cod-liver oil, which, in small doses of one or two teaspoonfuls three times a day, with or without malt extract, we have often known to be of great service. With the cod-liver oil, if desired, creosote (♏v. to ♏x.) may be combined.

The above are at best but palliative measures, which assist the patient in resisting the damp and fogs so associated with our climate, and to which the malady is in great part due. These can only be avoided by wintering in one or other of our brighter, warmer, and more sheltered health resorts, or by spending the season abroad. In England the health resorts upon the South Coast prove most beneficial, and among them we may number Penzance, Falmouth, Torquay, Sidmouth, Lyme Regis, Bournemouth, Ventnor, and Hastings, at all of which the climate is suitable. For those living in the Northern and Midland counties, Tenby, Colwyn Bay, and Llandudno may be recommended, since at these places the winter temperature is moderately warm and abundance of sunshine is experienced; at the two latter places the background of hills affords protection from the northern winds to which, from their geographical position, these stations are exposed. Grange and Southport, on the Lancashire seaboard, are also suitable. Of the inland resorts, Malvern, and Bridge of Allan are, perhaps, the best adapted to the treatment of the disease.

In Ireland, Rostrevor on Carlingford Lough, St. Ann's, Blarney, near Cork, or Glengariff, may be suggested.[13]

Should the patient be sent abroad, one or other of the health resorts on the French or Italian Riviera, such as Hyères, Cannes, Nice, Mentone, or San Remo, may be chosen. Of places farther south, Egypt was popular before the war, and, provided the patient avoid Cairo and stay at Mena House, Helwân, or Assuan, where the pure desert air, with its stimulating qualities, brilliant sunshine, and great dryness, may be enjoyed, no better selection could possibly be made

by those who can go so far. But whether the patient winter in Egypt or on the Riviera, he must remember that in neither case will it be wise for him to return to England until the beginning of May. Otherwise, the biting east winds of spring will not be avoided, an attack of acute bronchitis may follow, and all the benefit derived from wintering abroad be nullified. In summer a course of treatment at Mont Dore, or if the patient be gouty, at Ems, may in certain cases be taken with advantage.

Peculiar Forms of Bronchitis.

The varieties of bronchitis were classified by Laennec according to the amount and character of the expectoration. He thus recognised, in addition to the ordinary muco-purulent form, a type in which the secretion was peculiar and excessive, "pituitous catarrh," and another, in which the expectoration was very scanty, and to which he applied the name "catarrhe sec." To these we may add a third, the so-called "plastic bronchitis."

1. **Pituitous Catarrh. (Bronchorrhœa Serosa).**—This form of bronchitis was first described by Laennec. "Pituitous catarrh," he wrote,[14a] "is a catarrh in which the expectoration is colourless, transparent, stringy, frothy on the surface, and which, when the foam has been removed, resembles white of egg whipped up in water." Later he states that the flux is always abundant. To this description we may add that the characteristic sputum, or pituita, is *non-albuminous*, thus distinguishing it from the highly albuminous "serous expectoration," which is brought up in cases of acute œdema of the lung, and which resembles it exactly in naked-eye appearance (see pp. 114 and 355).

Pituitous expectoration occurs sometimes in cases of tuberculosis of the lung, whether miliary or otherwise, and in certain varieties of pulmonary cancer. In the cases which we are now considering the malady is a primary affection, unconnected with any pre-existing disease.

Primary pituitous catarrh may be acute or chronic, although both forms of the disease are excessively rare. In the acute form the patient, who may previously have been in perfect health, is suddenly seized with great oppression of the chest and cough, and dyspnœa rapidly ensues. Large quantities of

colourless frothy sputum (four pints in one instance referred to by Laennec) are brought up, and suffocation may be imminent, or may actually occur. More commonly, after some hours of intense distress, the symptoms gradually abate, and the patient recovers, though he remains liable to a second attack, possibly some months hence. The disease is of exceedingly rare occurrence, but has been known to attack people of all ages, even little children. Should the patient die, the autopsy reveals nothing but a slight reddening of the bronchial mucous membrane.

The chronic variety is less rare than the acute, and is most apt to occur in the aged, although adults are not free from attack. The chief symptom is the expectoration of large quantities of characteristic sputum, amounting possibly to four or six pints a day. The general health of the individual may not suffer for years, and as the great bulk of the sputum is sometimes brought up in a morning and evening attack, a fairly active life is not incompatible with the disease. In other cases, as in the following, recorded in his Lumleian Lectures by the late Dr. Gee,[12b] the patient is completely incapacitated:

"A Japanese, twenty-five years old, came over as a ship's steward to England, and immediately upon his arrival began to suffer from shortness of breath and cough: a new thing for him. After these symptoms had lasted for fourteen weeks, he was admitted to the hospital. There was much dyspnœa and most abundant expectoration of pituita. Tubercle bacilli were not found. The physical signs were those of bronchitis. For days together he seemed about to die from suffocation, but under very careful treatment he improved until, without any obvious cause, the symptoms recurred in full severity. Another improvement was followed by another relapse, and more than four months had passed away since his admission ere he could be discharged fairly convalescent. This was on May 8, but on June 16 he was readmitted, with all his former symptoms; he went out on July 16. On December 5 he was readmitted once more, and the time of the year was probably the reason why we could not get rid of him till February 25. He had now been a patient on and off for more than a year, and, as there seemed no end to this sort of thing, the Sister of the ward got up a small subscription and shipped him off to the Antipodes."

2. **Bronchitis Sicca (Catarrhe Sec).**—Dry catarrh, or, as it is better termed, "bronchitis sicca," was, like pituitous catarrh, first described by Laennec, and, according to him, consists

anatomically in a swelling of the mucous membrane of the smaller bronchi, with but little secretion. The swelling is sometimes so intense as to occlude the tubes, whilst in other cases the remaining lumen is obstructed by small semitransparent pearly-grey masses of secretion the size of a hemp or millet seed, to which Laennec gave the name of "crachats perlés" ("*sputa margarītacea*," pearly sputum).[14b]

Clinically, the disease is more often of the chronic than of the acute type, and produces at first but few symptoms, the patient being merely somewhat short of breath, and bringing up in the morning, with an occasional cough, a few pellets of pearly sputum. As the disease advances, the breath becomes shorter, the cough more troublesome, and gradually emphysema developes. Every now and then acute attacks are experienced. These last, perhaps, three or four days, during which the cough and dyspnœa are accentuated, and then, with the expectoration of an increased amount of the characteristic sputum, the symptoms gradually subside. Sometimes an acute attack occurs quite apart from any pre-existing disease, and by its sudden onset and marked dyspnœa gives rise to great anxiety.[15]

3. **Plastic Bronchitis.**—This disease is characterised by signs of bronchitis, the occurrence of a plastic exudation into the bronchial tubes, and the appearance in the sputum at recurring intervals of the casts so formed. Cases were observed by Galen and others of the early writers, but our actual knowledge of the malady dates from the appearance of Lebert's monograph in 1869.[16]

The disease is of very rare occurrence, and twice as common in the male as in the female. It may occur at any age from infancy to advanced life, but most of the recorded cases have commenced between the ages of fifteen and fifty.

Appearance and Nature of the Casts.—The branching casts, which we associate with the disease (Plate VIII.), are not seen as such in the sputum. When expectorated, they are generally rolled up, and appear as rounded masses covered with mucus, the true nature of which is apt to be overlooked, and it is not until they are floated out in water and carefully unravelled that they are proved to be moulds of the bronchial tubes. A perfect cast, extending sometimes to the most minute ramifications of the bronchial tree, may be as much

PLATE VIII

CASTS EXPECTORATED IN PLASTIC BRONCHITIS

The plate shows representations in actual size of the casts expectorated in this disease. That on the right, from a woman who attended the Brompton Hospital, illustrates the extension of the plastic inflammation into the finest bronchial tubes; that on the left, from a girl aged ten, a patient at St. Bartholomew's Hospital, shows the great size to which such casts may attain. The history of this second case, from the Museum Catalogue, is as follows:

"The girl had suffered from intermittent attacks of fibrinous bronchitis since her seventh year, the present attack being the seventh. The illness would commence suddenly with cough, headache, and malaise. After four or five days of such illness, which was attended by moderate fever and dyspnœa, she would expectorate casts like those seen in the specimen, with immediate relief of symptoms. The average duration of an attack was about four weeks. The physical signs before the discharge of a cast consisted of impaired percussion resonance over the area concerned, with abolition of breath and voice sounds, and displacement of the heart towards the affected side. Discharge of a cast temporarily restored the physical signs in the chest to the normal. The child left the hospital well some seven weeks after the onset of the present attack."

PLATE VIII

Casts expectorated in Plastic Bronchitis.

To face p. 196.

as seven inches long, but often they are smaller than this, and not infrequently are expectorated in separate little pieces. In appearance they are white or yellowish in colour, and are sometimes semi-transparent. The diameter of the casts varies, but at their maximum they are rarely larger than a goose-quill. On section, some prove to be solid; in others a lumen is found.

When examined under the microscope, the cast is seen to consist of an outer layer, with numerous concentric laminæ, amongst which leucocytes are found embedded. In some cases, the laminæ are seen to be formed into separate cylinders, suggesting "that the exudation starts in the fine bronchi, and is then gradually pushed up into the larger tubes" (Bettmann).[17a]

The chemical constitution of the casts has given rise to much discussion. It was long thought that they were composed chiefly of fibrin, and some undoubtedly are of this nature, the material staining by Weigert's method, and presenting the chemical characteristics of this substance. In other cases, though a few threads of fibrin may be present, the great mass consists, not of this material, but of mucin.[18] Since, therefore, in a given case the cast may prove to consist of either substance, the name "fibrinous bronchitis," often used to indicate the disease, is hardly applicable.

With regard to the site of formation of the casts within the bronchi, Bierman states that this may be inferred from the length of the intervals between successive points of bifurcation, and Dr. Ewart's researches into the structure of the bronchial tree would corroborate this. It is important to remember that the bronchi of the upper lobe are peculiar in presenting short and rapidly branching segments as compared with the elongated tubes of the lower lobe. The calibre of the casts would also be of assistance in deciding the question of site.

Ætiology.—The causation of the malady is still obscure. The few post-mortem examinations which have been recorded have revealed nothing beyond slight desquamation of the epithelium of the affected bronchi, together with enlargement of the mucous glands. Bacteriology has given but little assistance. In the casts pneumococci, streptococci, and staphylococci have been discovered, but, as they are also found

in simple bronchitis, their presence cannot explain the peculiar features of this variety. The Klebs-Löffler bacillus is never found, thus proving that the complaint has no connection with diphtheria.

In certain cases plastic bronchitis has occurred during the course of pulmonary tuberculosis or of morbus cordis. But neither of these diseases appears to possess a casual relationship, and at present the true pathology of the malady remains obscure. In some cases, as in one that has come under our notice, a gouty source will be found in associated arthritic phenomena. The malady is doubtless analogous in some respects with the mucous colitis with which we are more familiar.

Symptoms.—In the majority of cases the disease is associated with chronic bronchitis, which, having lasted for some months or years, is at last complicated by the occurrence of a plastic exudation. The cough becomes violent and as a rule paroxysmal; the dyspnœa may be extreme; and, finally, after some hours, relief is obtained by the expectoration of masses, which, on floating out in water, prove to be bronchial casts. After the attack the patient generally remains better for some days, though still coughing up fragments of casts, until gradually his breathing again becomes more oppressed, the cough more hard and paroxysmal, and a second attack is experienced, followed perhaps at intervals by others. With each attack a mild degree of pyrexia may be associated, and hæmoptysis sometimes precedes the appearance of the casts. After a time the patient recovers, though still remaining the victim of chronic bronchitis; but after years of apparent immunity, he will, perhaps, suffer a recurrence of the plastic attack. Indeed, the tendency of the disease to recur forms one of its most noticeable features.

In less common cases plastic symptoms may complicate an attack of acute bronchitis—the first from which the patient has suffered—but in other respects this variety of the disease, which is sometimes spoken of as acute, does not differ from the more chronic type already described.

Physical Signs.—The signs of the disease are very indefinite, being simply those of general bronchitis, with perhaps a certain degree of dulness, resulting from collapse, with very feeble breath-sounds over the area of bronchi affected. These signs may be heard over any part of the lungs, since any por-

tion of the bronchial tract may be affected, but they are more common at the base. In certain cases, when the cast has become loosened, but not yet expectorated, a coarse, dry, clicking sound—the so-called *ventil-geräusch* or *bruit de drapeau*—has been described as heard on auscultation, due, it is believed, to the flapping backwards and forwards of the free edge of the cast during respiration.[17b] But this sign is, in our experience, exceptional, and the recognition of the disease must rest on the symptoms rather than on the physical examination of the patient.

Diagnosis.—Diphtheria, poisoning with ammonia fumes, or exposure to superheated steam, may all produce a membranous exudation into the bronchial tubes; but in none is the disease likely to be confounded with the malady now under consideration. Again, after hæmoptysis semi-organised blood-casts are sometimes expectorated; but their reddish colour and the history of their occurrence explain their pathology. The possibility of a foreign body in the bronchus sometimes arises, and then requires a fresh scrutiny of the history of illness and special examinations, to which we shall hereafter refer (Chapter XIV). Plastic bronchitis, in fact, is not difficult to diagnose when its presence is suspected. When, therefore, in a given case a patient suffers from attacks of dyspnœa, which are relieved by the occurrence of expectoration, this should always be carefully examined for casts, any suspicious rounded mucoid masses being teased out and unravelled in water. If this were done more frequently, it is probable that the disease would be recognised as less rare than at present, since, in a certain number of cases in which casts have been discovered, the attacks of dyspnœa have been but slight, and by no means characteristic.

Prognosis.—As we have already stated, the tendency of plastic bronchitis is to recur, and by thus leading to emphysema, to curtail the duration of life. In the majority of cases the attack itself does not prove fatal; nevertheless, a study of the literature shows that the danger of an immediately fatal issue has been underestimated. When this occurs, it is due to suffocation from the dislodgment of an extensive cast, which becomes impacted in the larynx—an accident most to be feared in the very old or very young, in whom cough is least effective.[19]

Treatment.—During the attack our endeavour must be to assist the patient in expectorating the casts. To this end the air should be warmed and moistened by a steam-kettle, and, if dyspnœa be urgent and suffocation imminent, an emetic may be given. Iodide of potassium in moderate doses is of service in loosening the casts and in preventing their fresh formation, and a prolonged course is indicated in cases of a markedly recurrent character. It is possible that a vaccine prepared from the bronchial secretion might prove of value.

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CHAPTER XIII

NARROWING AND DILATATION OF THE BRONCHI

Narrowing of the Bronchi.—*General narrowing* of the bronchi is practically only met with as a consequence of swelling of the mucous membrane in catarrhal affections or exudative maladies such as plastic bronchitis and diphtheria. Doubtless cases may occur in which there is some general diminution in the calibre of the bronchial system, but they are not recognisable during life.

Localised narrowing of a bronchus may arise from: (1) Cicatricial contraction of an ulcerated surface within the bronchus; (2) contractile sclerosis of the bronchial sheath at one or more points; (3) invasion of the calibre of the bronchus by malignant growths; (4) pressure upon the bronchus by enlarged glands, growths, hydatid or aneurismal tumour. Let us now consider these causes in further detail.

(1) The cicatricial changes ensuing upon ulceration are amongst the very rare causes of bronchial narrowing, and are almost always of syphilitic origin.

(2) In association with the more chronic indurative form of phthisis, it is not uncommon to find at certain points bronchial tubes narrowed or even obliterated by what may be regarded as cicatricial growths involving the sheath of the bronchus. This change may occur at points adjacent to tuberculo-fibroid nodules, or at the entrance to cavities which have undergone considerable or complete contraction and are surrounded by a zone of cicatricial induration in which the entering bronchus is involved. In this way partially contracted cavities are not infrequently closed. Sometimes when a cavity becomes thus shut off by occlusion of its communicating bronchus, the purulent contents subsequently inspissate into a creamy débris, which at a later period becomes cretaceous. In other cases the purulent secretions

increase, and, becoming pent up, cause elevation of temperature and other signs of abscess; finally, when they attain to a certain degree of pressure, the narrowed bronchus yields, and a discharge takes place, after which accumulation again commences.

(3) and (4) The invasion of malignant growths and the pressure of tumours are amongst the most common causes of obstruction to the main bronchi; and are of too obvious a mechanism to require further exposition.

The necessary consequences of narrowing of a bronchus at any point are (*a*) more or less complete retention of secretion behind the obstruction; (*b*) variable changes of a destructive kind in the lung itself.

In cases in which the secretion is derived from the bronchial mucous membrane, the lung, not being as yet involved, it is thick, viscid, and muco-purulent, and collects in such quantities as to distend the bronchi behind the obstruction. When sufficient distension has taken place, a paroxysmal cough will expel through the narrowed orifice a certain portion, the overflow, so to speak, of the collection, in the form of thick, viscid, and more or less nummulated sputa, generally not offensive.

The condition of lung that attends narrowing of a bronchus is in the later stages one of airless collapse, to be followed shortly by more or less thickening and fibrosis of the pulmonary structure, with dilatation of the tubes behind the stricture. As contended by the late Dr. Pearson Irvine,[1] and more recently, in his Bradshaw lecture by Dr. Newton Pitt,[2] dilatation of the affected lung, sometimes even sufficient to suggest pneumothorax, may be the primary consequence of pressure upon the bronchus, the effect of the narrowing being to impede expiration, whilst inspiratory effort is successful. Such primary dilatation is, however, but rarely seen, and it is probable that so soon as secretion begins to collect behind the obstruction, the air-cells can no longer be penetrated by air, whilst that which remains in them must slowly be expelled or absorbed. Secondary changes of a destructive character not infrequently ensue in a lung, the main bronchus of which is thus occluded by pressure, changes which were thought by the late Sir William Gull[3] to arise from disturbed innervation through pressure upon the pulmonary plexuses. But

there can be little doubt that in the great majority of cases we must trace these changes, not to any nervous influence, but to the retention of secretions which necessarily become more or less septic—a condition analogous to that met with in the kidney in old-standing hydro- or pyonephrosis, and also in other obstructive urinary diseases.

The Symptoms and Signs of bronchial narrowing need only be considered with regard to those cases in which the constriction is situated at one or other main bronchus. In these cases the signs of narrowing of the bronchus sometimes present themselves before the cause of the narrowing can be precisely made out.

Relatively feeble breath-sounds of harsh or blowing quality over one lung, most frequently in the upper interscapular region of the affected side, are the first signs to be observed. The percussion note, at first unaltered, becomes of higher tone over the affected area, the vocal fremitus and resonance being at the same time lessened. The exaggeration of the vesicular breathing on the healthy side is in marked contrast with its feebleness and want of vesicularity on the opposite side.

Soon a further sign, stridor, makes its appearance. At first slight, and only heard occasionally, it gradually becomes more marked, until even when at rest each inspiration, and to a less degree each expiration, is attended with a coarse, stridorous noise. Sometimes, too, we may notice recession over the affected side, a sign which, in conjunction with the preceding, is most suggestive. As the case proceeds, the feebleness of the breath-sound becomes increased to final extinction, while the diminished mobility and shrinking of the side, with impaired percussion note, becomes more definite.

From the first there is cough, which may be of laryngeal type, from involvement of the nerves of the larynx concurrently with the bronchial compression. The cough is, moreover, generally spasmodic in character, the passage of the thick sputa through the stricture giving rise always to more or less, sometimes to very severe, paroxysms of dyspnœa. As pulmonary collapse proceeds, pleuritic pains and the signs of dry pleurisy present themselves, and, with advancing dulness, may lead the observer away from the right diagnosis. The heart is uncovered and shifted towards the affected side,

unless its position be otherwise determined by the pressure of a tumour.

In the most common class of cases, those in which the narrowing is due to the compression of an aneurism or malignant growth, other signs of pressure, ocular, laryngeal, and so forth, will as a rule be present, thus throwing helpful light upon the nature of the affection. It is in cases of this kind also that assistance may be looked for from an X-ray examination, a definite pulsating aneurismal tumour, for instance, being sometimes revealed, when from the physical signs alone it would have been impossible to arrive at a certain diagnosis.

In cases of syphilitic narrowing there will be the history of syphilis, the signs of narrowing, and the absence of those of tumour. In an interesting case that has come under our notice, in which the left bronchus was almost obliterated, and the lower portion of the trachea narrowed by syphilitic cicatrices, in addition to an absence of the signs of aneurism, it was observed that the patient's distress was greatly increased by an attempt to lie in the prone position, and that he felt most easy when erect and walking about the room. In aneurism the patient's troubles are much increased by movement, whilst the prone position sometimes gives ease from the falling forward of the aneurism and the consequent relief of pressure.

Treatment.—The cause of the constriction, be it aneurism or syphilis, must be appropriately treated; and in most cases much may be done in the way of giving ease.

Regulated doses of chloroform for inhalation are valuable in relieving spasmodic cough and in aiding expectoration. For this purpose a few drops of chloroform at a time may be placed upon absorbent cotton-wool in a small bottle to be sniffed. We have also known a pungent vapour, such as strong peppermint essence in hot water inhaled through the nostrils, to give relief. In cases in which the paroxysmal dyspnœa arises from compression of the bronchus by aneurism, nitro-glycerine (1 minim of the liquor trinitrini) will assist by lessening tension within the sac. Expectorant remedies are worse than useless. Chloral and paraldehyde give more promise of usefulness than opiates. Indeed, chloral in small repeated doses is a most valuable drug in lessening

the spasm which always intensifies the symptoms in cases of narrowing of the main bronchus, thus giving time for other remedies, such as iodide of potassium and mercury, to take effect.

When the main bronchus is involved the prognosis is of the gravest character.

Dilatation of the Bronchi—Bronchiectasis.—This affection consists in a manifest widening of the bronchial tubes over a more or less limited area. It is by no means a common disease, and occurs much more frequently in males than in females. It is more rare among the well-to-do than among the poor.

No age is exempt from it, but, as indicated by the following statistics, compiled from the records of the Brompton Hospital by Dr. D. Barty King, it is most common between the ages of ten and forty. We must note, however, that in this table the incidence of the disease in the first decade of life is probably underestimated, owing to the fact that the number of children admitted into the Brompton Hospital is limited.

TABLE SHOWING THE AGE-INCIDENCE IN SIXTY CASES OF BRONCHIECTASIS FROM ALL CAUSES.[4]

Age.	Number of Cases.	Per Cent.
0–10	1	1·6
10–20	14	23·3
20–30	15	25·0
30–40	16	26·6
40–50	8	13·4
50–60	4	6·7
60–70	2	3·4
Total ...	60	100·0

MORBID ANATOMY.—Anatomically, the disease appears in two chief forms: the *cylindrical* and the *sacculated.* In cylindrical or fusiform bronchiectasis the dilatation involves some length of the tubes, varying from a few inches to a system of tubes ramifying through an entire lung. The enlargement is uniform throughout the length of tubes affected, though sometimes at the distal end of the tube there may be a further ampullary dilatation. In sacculated

PLATE IX

CYLINDRICAL BRONCHIECTASIS

The specimen shows the left lung cut open and the sides turned back. On the left hand the specimen has been untouched; on the right the bronchi have been opened up to show the general cylindrical dilatation. On examining the left-hand portion, the lung is seen to present the appearance of being filled with ovoid cavities, which varied in size from a bean to a bantam's egg. An examination of the right-hand portion, however, shows that these apparently separate cavities are in reality sections cut at varying angles of the bronchi, which throughout their course are dilated in a cylindrical manner, with in some cases a further terminal expansion. Between the dilated bronchi the lung tissue has undergone fibrosis. The pleura is everywhere adherent, but not much thickened.

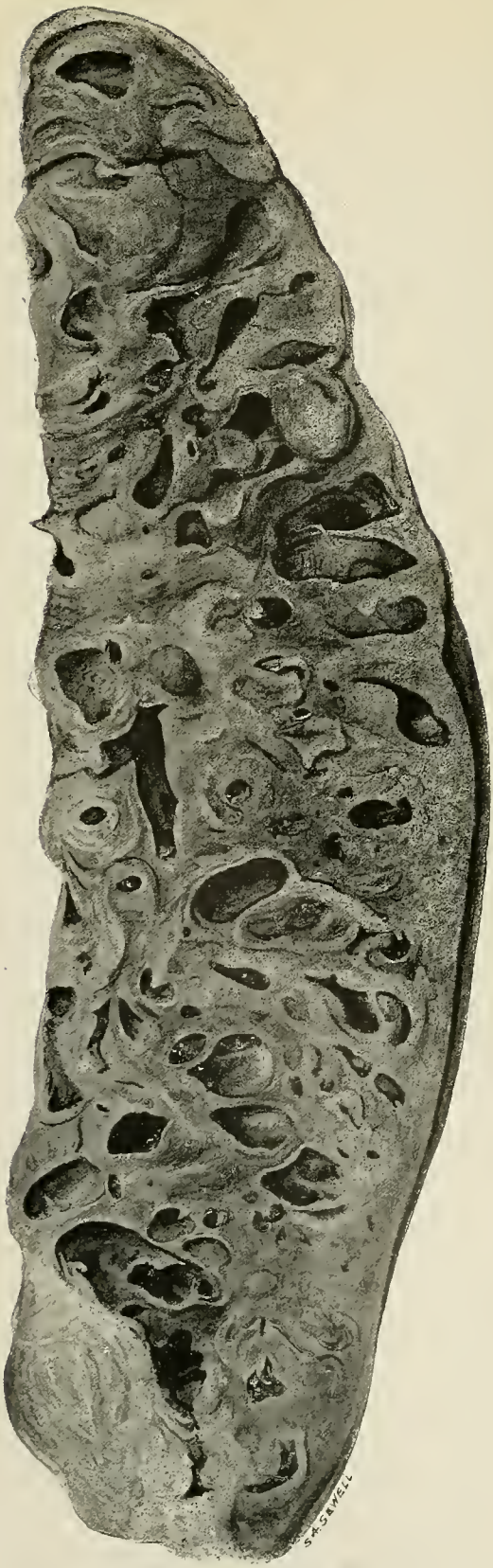
From a man aged twenty-one, who suffered from bronchiectasis with much cough and fœtid expectoration, and who died from an abscess in the right frontal lobe. The right lung contained some patches of recent broncho-pneumonia, but the bronchi were not dilated. The spleen showed lardaceous degeneration.

(From the Brompton Hospital Museum. $\frac{9}{16}$ natural size.)

Cylindrical Bronchiectasis.

To face p. 206.

PLATE X



SACCULAR BRONCHIECTASIS.

To face p. 207.

SACCULAR BRONCHIECTASIS

The drawing shows a portion of the left lung in a condition of advanced saccular bronchiectasis. The lung is shrunken, and its substance has given place to smooth-walled cavities of various size, separated from each other by trabeculæ of fibrous tissue, in which, under the microscope, a few pulmonary alveoli may still here and there be detected. The pleural layers are adherent. Microscopical examination showed the cavities to be formed by dilatation of the bronchi and bronchioles; their walls were lined by granulation tissue, with no trace of epithelium remaining. The right lung was emphysematous, and contained in its lower lobe a few cavities similar to those on the opposite side. The heart was dilated and drawn over to the left.

From a man aged twenty-eight, who died of heart failure, after suffering for twelve months from shortness of breath, and for three weeks from dropsy.

(From the Museum of St. Bartholomew's Hospital. $\frac{7}{8}$ natural size.)

PLATE X

bronchiectasis a restricted portion of a tube is enlarged to a globular form, from a quarter of an inch to an inch in diameter. The whole calibre of the tube is generally involved, and the dilatation may be solitary or there may be many scattered through the lung. Small openings lead from the rounded and apparently closed distal side of the sacs to fine tubes, the branchlets of the widened bronchus. The illustrations (Plates IX. and X.) show the naked-eye appearances presented by these two varieties of the disease.

Of the two forms, the cylindrical is by far the more common, though it may be combined with a certain degree of saccular dilatation. It is probable, indeed, that not a few of the cases which have been described as saccular have been in reality examples of the cylindrical form in which the tubes have been cut at varying angles, thus giving in a section of the lung the appearance of more or less rounded cavities of different size. In such cases it is only by slitting up the bronchi throughout their length that the true nature of the case becomes apparent (see Plate IX). In other instances, again, in which a bronchus does open directly into a large sacculated cavity, we must hesitate before assuming that the cavity in question is necessarily of simple bronchial origin, even though there may be definite evidence elsewhere of dilatation of the tubes. Not uncommonly it is due, in part at least, to gangrene, whereby the walls of adjacent bronchiectatic cavities are destroyed, thus producing extensive and irregular excavations. These sometimes closely resemble true pulmonary vomicæ. The distinction is made post-mortem by finding in the cavity walls remnants of cartilage, which reveal the original nature of the dilatation.

Bronchiectasis, whether cylindrical or saccular in nature, may be met with in any area of the lung, and in any lobe; but it is most common in the lower lobe, since here retention of secretion, a potent factor in the ætiology of the disease, is more liable to occur than in the upper lobes, where gravitation assists in emptying the tubes. Out of fourteen cases of simple bronchiectasis (unassociated with tubercle) in which the autopsy was made by one of us,[5] in two only was the disease limited to the upper lobe; in a third it was most marked in this situation, though affecting other lobes as well. In the remainder the disease had apparently commenced, as

usual, at the base of the lung. But at the time of death, the condition is not commonly limited to the lobe first affected. Thus, if we add to the 35 cases from the Brompton records quoted by Sir J. Kingston Fowler,[6a] the 14 more recent ones referred to above, we obtain a total of 49 autopsies. Analysing these, we find that in 31 cases (63 per cent.) both lungs were affected at the time of death, in 18 (37 per cent.) one lung only; in only 12 cases (24·5 per cent.) was the lesion limited to one lobe. We may add that when the disease passes from one lung to affect the other, it is again the lower lobe which is usually first affected, the tendency of the disease being to extend from below upwards.

ÆTIOLOGY.—If we exclude certain rare cases of congenital origin, which are probably to be regarded as malformations or errors of development, it may be said that bronchiectasis is never a primary disease. It is dependent upon either increased pressure within the tubes or traction upon their walls, a necessary phase in all cases being some structural change in the bronchial tissues.

The causes may be classified under three headings: (1) bronchial; (2) pulmonary; and (3) pleural; the relative importance of which may be gauged from the following table based upon the records of the Brompton Hospital, which we have taken from Dr. Acland's paper.[4]

TABLE SHOWING THE CAUSES OR ANTECEDENT CONDITIONS, AS FAR AS COULD BE ASCERTAINED, IN FORTY CASES OF BRONCHIECTASIS, VERIFIED BY POST-MORTEM EXAMINATION.

Disease.	Number of Cases.	Percentage.
1. Chronic bronchitis	18	60·0 (77·5)
Chronic cough since childhood ...	6	
Bronchial stenosis—		
Tumour	3	17·5 (77·5)
Aneurism	2	
Foreign body	2	
2. Pneumonia	4	10·0
3. Pleurisy	5	12·5

Let us now consider the various causes in more detail.

1. *Bronchial.*—From the table which we have just given, it will be evident that affections of the bronchi play by far

the most important rôle in the ætiology of bronchiectasis. "Chronic bronchitis" and "chronic cough" account for more than half (60 per cent.) of the cases quoted, and bronchial stenosis is responsible for another 17·5 per cent., making a total of 77·5 per cent., thus leaving only 22 per cent. to be accounted for by pneumonia and pleurisy.

In regard to the relation of chronic bronchitis to bronchiectasis, there can be little doubt that the disease is determined by two factors: (*a*) changes of an inflammatory kind occurring in the deeper layers of the bronchial mucous membrane, to which we have referred in the chapter on bronchitis; (*b*) the rending force of the cough distending the tubes, the elasticity of which has already become permanently impaired. It is the state of nutrition of the walls of the bronchial tubes which is the determining factor as to whether they will yield or not under the stressful conditions of chronic cough. In cases in which the texture of the walls has been damaged by inflammatory change bronchiectasis is apt to occur, and it is in this regard that any condition involving retention of secretion has so damaging an influence. Thickening of the surrounding lung tissue which to some extent accompanies the dilatation proceeds further after the bronchiectasis is established.

The inhalation of a *foreign body* into a bronchus is not of common occurrence, hence bronchiectasis is not often produced in this way. But the possibility that a foreign body may be at the root of the malady must be borne in mind in every case, since its early detection and removal by *bronchoscopy* may be the means of curing the patient, or at least preventing the occurrence of more than a mild degree of bronchial dilatation. As we shall point out in the succeeding chapter (see p. 232), the symptoms due to the inhalation of the foreign body may be slight, and therefore in every case of bronchiectasis careful inquiry must be made as to any history suggestive of such an occurrence. In the museum of the Brompton Hospital may be seen specimens of bronchiectasis due to such different matters as a molar tooth, an ear of corn, and in two cases a piece of bone, in one of which there was no history of the inhalation.

It is interesting to note that a tumour growing from the interior of the bronchus and partially occluding it may act

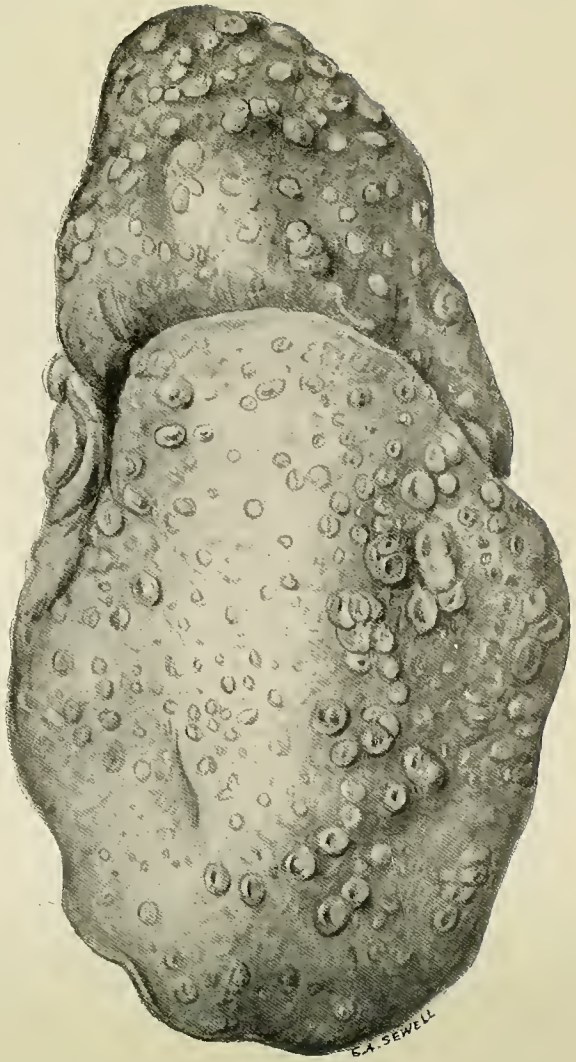
like a foreign body and lead to bronchiectasis, though such primary growths are of great rarity.[7]

2 and 3. *Pulmonary and Pleural.*—When the vesicular texture of a lung is obliterated by permanent collapse or fibrous growth, the tendency of the inspiratory force is to widen the bronchial tubes which traverse that lung; and as the condensed tissues in which the tubes are embedded further contract, the widening becomes more marked. Secondary to a pneumonia more often broncho-pneumonic in character, or a dry pleurisy, a portion of a lung, perhaps a whole lobe, will thus in certain cases become condensed by connective-tissue growth, which extends from the pleura along the interlobular septa and from the sheaths of the bronchi and vessels. The fibrous overgrowth thus produced has been termed chronic interstitial pneumonia (see Chapter XIX.). The pleural surfaces under these conditions are almost invariably adherent, and the lung being held attached at its circumference by the adhesions, and its bronchial tubes fixed at the root, it is clear that when the thoracic wall has receded, and the surrounding organs, thoracic and abdominal, have approximated as far as possible, any further contraction of the lung must drag upon the bronchial tubes and extend their calibre in all directions. As the result, the medium and subcapillary tubes embedded in the depth of the lung become widened out into fusiform and globular cavities, the finest tubes becoming extensively obliterated in the course of the contractile disease.

In cases of thoracentesis for old-standing empyemata, bronchiectasis amounting to complete loculation of lung may arise in a manner not essentially different from that just described. In such cases obliteration of the pleural cavity is finally effected from above downwards by the gradual growth of adhesions agglutinating the pleural surfaces. The parenchyma of the lung is, however, in these cases irretrievably condensed, so that the degree of enlargement of the organ necessary to fill the remaining thoracic space is only to be obtained by widening out the bronchial tubes, and further contraction of the fibrous lung must continue to have the same effect. Thus the lung in some cases comes to be a mere shell, enfolding bulbous bronchial cavities.

In congenital collapse of any portion of the lungs—*atelec-*

PLATE XI



Acute Bronchiolectasis, showing the Honeycombed Appearance of the Lung.

ACUTE BRONCHIOLECTASIS

(*The "Honeycomb Lung"*)

The drawings show the appearance of the lung. On the outer surface numerous bullæ are seen, while the cut section presents a general honeycombed appearance, due to the presence of innumerable small cavities. Microscopical examination proved that these were dilated bronchioles, with inflamed and infiltrated walls. There was a marked degree of acute emphysema, but no evidence of tubercle.

From a child aged three and a half years, who died of acute bronchitis after an illness of only twelve days.

(From the Museum of St. Bartholomew's Hospital.
Natural size.)

PLATE XI

tasis pulmonum—the bronchial tubes ramifying through that portion are widened.

Bronchiectasis having been induced as a result of one of the causes described above, certain further changes now occur in the dilated tubes. The secretions in them tend to accumulate, partly from the altered shape of the tubes and the destruction of their ciliated epithelium, and partly owing to the hardened tissue, by which they are surrounded, rendering effectual expulsion with cough impossible. Decomposition of the stagnant secretion generally follows, resulting in septic inflammation of the cavity walls and of the adjacent parts. It is in this stage that neighbouring bronchiectatic cavities may coalesce from softening or gangrene of their walls, and thus give rise to the irregular excavations of the lung to which we have referred.

We have spoken hitherto of chronic bronchitis as a cause of bronchiectasis, but we must add that in young children acute bronchitis may also lead to dilatation of the tubes. In this case, however, as shown by Dr. Sharkey[8] and others, the dilatation commonly affects not the larger bronchi, but the bronchioles, giving to the lung a curious worm-eaten appearance—the so-called "*honeycomb lung*"—owing to the innumerable small saccular cavities distributed throughout (Plate XI.). These also appear on the surface of the organ as small round transparent-looking vesicles. This variety of the disease, in its marked stages at least, is not common, and is of little importance clinically, since it cannot be distinguished from the antecedent bronchitis. From its somewhat special features it has been named "*acute bronchiectasis*" or "*bronchiolectasis*." It is possible that there may be a recovery, partial at least, from this acute dilatation of the tubes.

Symptomatology.—The symptoms of bronchiectasis differ much in different cases, and may, indeed, be entirely absent. Thus at the autopsy of a patient who died of acute bronchitis we have found marked cylindrical dilatation of the bronchi in the right upper lobe, though there had been no symptoms referable to this during life, and though the disease had evidently been of some standing. No doubt the site of the affection and the good drainage afforded to the tubes were responsible for this immunity from symptoms.

As a rule, the patient is not so fortunate, and symptoms manifest themselves, at first not very serious, but gradually becoming more urgent. One of the earliest noticed is *cough*, with attendant *expectoration*. This is at first muco-purulent in character, and not great in amount. As the disease advances, the cough increases, and with it the amount of sputum, until at last large quantities—a pint or more in the twenty-four hours—may be expectorated. The manner of its expulsion is often characteristic. The patient may not expectorate for some hours, and then perhaps, after some movement which disturbs the contents of the over-full tubes, he will begin to cough, and with more or less violent paroxysms, frequently attended with retching, will rapidly bring up several ounces. We have even seen the expectoration discharged through nose as well as mouth, so severe and distressing may be the paroxysms. Such attacks may occur at any time, but are most frequently observed in the early morning, on first waking, the secretion having accumulated in the tubes during the hours of sleep.

The sputum, though becoming more abundant, may for long remain untainted; but sooner or later it becomes fœtid, and is often extremely offensive. Whiffs of foul gas frequently precede and attend the expectoration. In a case recorded by Trousseau[9] "the smell of the patient's breath was such as to render pestiferous the whole of the suite of rooms occupied by him, and even the staircase leading to them was redolent of the same stench;" and this is no highly-coloured description of what is sometimes observed. Although, however, the fœtor of the sputa when first brought up may be so great, it is curiously evanescent, owing to the volatile character of the substance to which the odour is due.

If placed in a glass and allowed to stand for a few hours, the sputum will be found to resolve itself into three layers—an upper frothy scum, a greenish watery layer, and at the bottom of the tube a grey purulent deposit, containing pus cells, Charcot-Leyden crystals, crystals of fatty acids, together with numerous micro-organisms, and often horribly offensive small yellowish masses—the so-called "Dittrich's plugs."

In spite of the distressing symptoms, the general health of the patient long remains surprisingly good. The features

PLATE XII

PULMONARY OSTEO-ARTHROPATHY.

To face p. 213.

The X-ray photograph of the right arm and wrist reveals the layers of new bone which are formed beneath the periosteum in this disease. They are visible on the shafts of the lower third of the radius and ulna, and along the shafts of the metacarpal bones. To a less degree they were also present on the first and second phalanges.

From a male patient, G. V., aged thirty-one, a compositor, the appearance of whose hands and wrists are shown in Fig. 26. He suffered from bronchiectasis originating in chronic bronchitis, and brought up as much as 197 ounces of offensive sputum during the week. When admitted into the Brompton Hospital in 1902, the right lung was found to be extensively affected, and the left to some extent also involved. The fingers were markedly clubbed, and the lower ends of both forearms and of both legs above the ankles were enlarged. The knee and ankle joints were somewhat swollen and tender, and contained fluid. X-ray examination showed that a layer of new bone surrounded the shafts of the lower third of the radius and ulna, and of the tibia and fibula on either side, and was also present on the metacarpals and the metatarsals, and to a less extent on the phalanges. The sputum was examined on many occasions for tubercle bacilli, but none were found. There was no evidence or history of syphilis.

After staying eight months in the hospital, he left but little improved; but shortly afterwards his cough and expectoration abated greatly, and when seen in July, 1904, he was only bringing up a very little inoffensive sputum. The enlargement of his bones was less marked, and X-ray examination showed that the deposits of new bone were thinner, though denser, than before. He died at home about two years later.

PLATE XII

are somewhat puffed and suffused, and there may be some slight lividity of colouring in lips and cheeks, the *facies* thus differing much from that often presented by the emaciated sufferer from tuberculosis. For some time there may be no pyrexia, and except for a little dyspnœa on exertion and the fœtor of the breath and expectoration, there would be nothing to prevent the patient from following his ordinary avocation.

At this stage *clubbing* of the fingers and toes makes its appearance, and in certain cases also a bulbous condition of the nose. The clubbing sometimes reaches a high degree, and becomes a point of some importance in diagnosis, though we have known cases of bronchiectasis in which it has been but little marked. Signs of *pulmonary osteo-arthropathy* may also in some cases be observed. The subperiosteal deposition of new bone is chiefly noticeable in the neighbourhood of the wrists and ankles—the forearms and legs, also the metacarpal and metatarsal bones, being the parts commonly involved. The changes, which are clearly apparent in an X-ray photograph (Plate XII.), lead to an enlargement with some tenderness of the affected areas (Fig. 26). Hæmoptysis is not uncommon, and copious and even fatal attacks have been known to result from rupture of an aneurism of a branch of the pulmonary artery traversing the wall of the ectasia.

Such are the symptoms during what we may call the second stage of the disease. Sooner or later constitutional symptoms make themselves apparent in the form of recurrent attacks of irregular fever, lasting perhaps a fortnight or more, and probably produced by septic absorption from the fœtid contents of the dilated tubes. These undermine the patient's strength, but he rarely wastes, as in a case of phthisis. Finally the end is ushered in by the advent of septic broncho-pneumonia, or perhaps by the onset of cerebral (or cerebellar) abscess. So well known, indeed, is the occurrence of this latter complication that it should be a rule at once to suspect it, if any cerebral symptoms develope in a patient suffering from bronchiectasis. Septic absorption and diffuse general bronchitis are less common terminations of the disease.

In cases in which the bronchial dilatation is secondary to pressure or contraction of a main air tube, the expectoration is often very viscid and mucoid, and not as a rule fœtid. It is expelled with considerable difficulty in successive thick masses

after violent attacks of coughing, at such times as there shall have been sufficient accumulation to force the narrowed passage.

FIG. 26.—PHOTOGRAPH SHOWING THE MARKED CLUBBING OF THE FINGERS, AND ENLARGEMENT OF THE WRISTS AND ADJOINING PARTS CHARACTERISTIC OF PULMONARY OSTEO-ARTHROPATHY.

(From a male patient, G. V., aged 31, who suffered from bronchiectasis.)

PHYSICAL SIGNS.—These, like the symptoms, vary in different cases, and no special sign can be said to be pathognomonic of the malady.

In bronchitic cases we may find at first merely a few sharp and persistent clicks at the base of one lung. As the disease progresses, and as fibrosis of the pulmonary tissue advances, the note over this region becomes gradually flattened and impaired, and the chest a little retracted. Later the heart is displaced towards the affected side. Signs of a cavity will now be discovered—bronchial breathing, bronchophony, and whispering pectoriloquy—which may suggest to the ear a single sacculated cavity, but which experience recognises as in all probability significant of several adjacent dilated tubes, with intervening lung converted into fibrous tissue. The situation of these signs will depend on the position of the cavity, but they are often best heard near the angle of the scapula or in the adjacent region of the axilla. Sometimes the "veiled puff" of Skoda may be heard, in which the inspiratory murmur, at first indistinct, becomes, as inspiration deepens, suddenly loud and bronchial. It is probably produced by the sudden removal of some obstruction in the tube communicating with a cavity,[10] and cannot therefore be regarded as pathognomonic of bronchiectasis.

In the signs themselves, therefore, there is nothing peculiar, but their variability and the way in which they alter, being now pronounced and now obscured, according as the bronchi are filled or empty, is a point which should be noticed. Such rapid alteration, if observed, would strongly suggest bronchiectasis, for no other cavities possess such facilities for speedy evacuation and refilling.

In cases in which neighbouring dilatations have coalesced, a considerable area of excavation may be produced, and we have even known almost the whole of the lower lobe to be converted into one large, evil-smelling cavity. In such cases cavernous breath-sounds and gurgling râles, with pectoriloquy, may be distinctly heard, and the percussion note acquires a perceptibly tubular character. The symptoms under these circumstances become more severe and hectic, nutrition suffers, and diarrhœa, with red tongue, is observed. Elastic tissue is now to be found in the sputum, but not tubercle bacilli, unless, as sometimes happens, there be an associated tuberculous infection.

Duration of the Disease.—Bronchiectasis, owing to its anatomical characters, is not a disease from which we may

expect recovery. At the best, all that we may hope for is that, by preventing decomposition of the sputum, we may be able to arrest its further progress. In nearly all cases, in spite of treatment, the disease gradually advances, although its duration varies much in different individuals, in some cases being exceedingly chronic, in others running a comparatively acute course.

Diagnosis.—*Basal phthisis* may sometimes be mistaken for bronchiectasis, especially in the early stages, when the upper part of the lung is not yet attacked. Basal phthisis, however, is rare; nevertheless its occurrence will serve to remind us that no case can be diagnosed with certainty as one of bronchiectasis unless repeated examinations of the sputum by the most careful methods have proved the absence of tubercle bacilli. It will, of course, be borne in mind that tubercle may become engrafted upon bronchiectasis, although this is uncommon.

A more important question is the diagnosis between bronchiectasis and a *localised empyema* rupturing into a bronchus. The two diseases have many features in common, and the diagnosis is sometimes a matter of great difficulty, especially when the empyema is small and at the diaphragmatic surface of the lung. The history of the case is important, especially the gradual appearance and laminated character of the expectoration, in contrast with its sudden and purulent outburst in empyema. A normal leucocyte blood-count will point strongly against suppurative pleurisy, but a leucocytosis is of less diagnostic value, being sometimes also present in bronchiectasis. An X-ray examination will occasionally indicate the presence of one or more cavities at the base of the affected lung, and thus aid in diagnosis, but as a rule, the thickening of the lung tissue around the dilated tubes leads to the formation of a general basal shadow, which is in no way distinctive.

As we have already indicated, a *foreign body* may be the cause of the bronchiectasis. A patient once came before one of us in the outpatient room at the Brompton Hospital complaining of cough of several weeks' duration. Examination revealed only a few crepitations, with slightly impaired note at one base. The diagnosis of tubercle or bronchiectasis was discussed, but before the former could be finally excluded the

patient removed all doubt as to the nature of the disease by coughing up the vertebra of a rabbit. Recovery ensued. The case illustrates the necessity of remembering the possibility of such an occurrence, even though no history of it be volunteered.

TREATMENT.—In considering the treatment of bronchiectasis our first duty is to exclude as far as possible the presence of a foreign body. If there be the slightest reason to suspect this, a bronchoscopic and X-ray examination must be made, and if the body be discovered an attempt made to extract it, which will often be successful. This subject is more fully discussed in the next chapter. When the foreign body is thus early removed, recovery or great amelioration of symptoms will ensue.

In other cases, and we fear they are the great majority, though we may not be able to cure the disease, yet considerable alleviation can at least be given to the patient by appropriate treatment.

The chief symptom of which complaint is made is the offensive character of the breath and of the sputum. This may be best combated by means of the creosote vapour bath, originally suggested by Dr. Arnold Chaplin.[11] To carry out this treatment a bare room is required, with a concrete floor if possible, and without any furniture or hangings which would be damaged by the creosote vapour. On the floor stands an iron tripod, supporting a metal dish or pan, into which commercial creosote is poured. Under the dish a Bunsen burner or spirit-lamp is placed for the purpose of volatilising the creosote, care being taken not to use too strong a flame, lest the creosote catch light. Should this accident occur, the flame may easily be put out by means of a little sand, which it is well to keep always in readiness. Both tripod and spirit-lamp are best placed on a metal tray, in case the evaporating dish should be overturned. When all is ready, the patient enters the room. He should be clad in a linen overall, and should be provided with a piece of lint or a handkerchief to protect his eyes. The Bunsen burner is then lit, and the fumes of the creosote begin to rise. The irritating effect of the vapour is soon felt by the patient, who commences to cough, and in this way succeeds in bringing up a large quantity of phlegm, to a great extent emptying his tubes. The

special action of the creosote now comes into play, the vapour being enabled to reach and act upon areas of the bronchial tract, which, having been freed from secretion, are in a condition to benefit by the cleansing property of the antiseptic.

Treatment should be commenced by a "bath" every other day, the patient remaining in the vapour for a few minutes only. Very soon a daily bath may be given of half an hour's duration, or longer, for a course of five or six weeks, and afterwards at gradually increasing intervals.

As a result of this treatment it is often found that a very sensible diminution in the fœtor of the sputum results, while sometimes the odour entirely disappears. Hand-in-hand with the improvement, a progressive diminution in the amount of the sputum may be observed, as in an instructive case recorded by Sir J. Kingston Fowler.[6b] In other cases, though the fœtor may diminish or disappear, the quantity of sputum remains the same. In others, again, the treatment seems to fail.

Should it not be found possible to commence the creosote baths at once, we would recommend as a palliative the use of some antiseptic inhalation, given in a Coghill's respirator. The following formula, long in use at St. Bartholomew's Hospital, is a useful one: Creosoti, tincturæ iodimitis, acidi carbolici, ætheris, and spiritûs vini rectificati, āā ʒi. The inhalation may be practised several times a day, and often does good in diminishing fœtor of the sputum.

Many cases of bronchiectasis can be kept fairly drained for years by the simple expedient of devoting half an hour two or three times a day to expectoration, by leaning over with head low, so as to allow any accumulated fluid to gravitate towards the area of glottic sensitiveness.

Internally, the administration of creosote, or of such drugs as turpentine, oil of sandal wood, and copaiba, the volatile oils of which are excreted by the bronchial mucous membrane, does some good, but the amelioration produced is comparatively slight. We have in some cases prescribed with advantage garlic, either the clove itself or the Syrupus Allii Aceticus, as suggested by the late Dr. Vivian Poore.[12] Some patients bear the drug well, but others resent the penetrating odour of garlic which becomes imparted to the breath.

Another plan of treatment, recommended by the late Sir T.

Grainger Stewart,[13] Mr. Colin Campbell,[14] and others, consists in injecting various solutions into the trachea through the larynx by means of a special syringe. The following formula was used by Grainger Stewart[13] for such intralaryngeal injections:

Menthol	10 parts.
Guaiacol	2 ,,
Olive oil	88 ,,

One drachm to be injected once or twice daily.

We have tried this and other solutions, and have found benefit to result in certain cases, the fœtor of the sputum sensibly diminishing under the treatment. A little practice will enable the operator to guide the syringe over the epiglottis and through the vocal cords before making the injection.

Should the excessive quantity of the sputum be a source of trouble and annoyance to the patient, an attempt may be made to lessen it by diminishing the daily quantity of liquid taken.

In some cases, where the absence of adhesions permits it, the performance of an *artificial pneumothorax*, and the compression of the lung by the introduction of nitrogen, as described in a later chapter, gives considerable relief. In this way the bronchi are compressed, and the accumulation of secretion and the absorption of toxines prevented. The relief is, however, not permanent, and can only be maintained by keeping up the positive pressure by the reintroduction of nitrogen at intervals varying from two to four weeks. Nevertheless, as a means of giving temporary relief, the method deserves consideration.

Surgical Treatment.—The condition of a patient suffering from bronchiectasis is sometimes so pitiable that it is not surprising that attempts have been made to give relief by surgical intervention.

Opening and draining of the cavity was first attempted, especially in cases in which the signs apparently pointed to a single large cavity at the base of the lung. In performing the operation, which is carried out as when dealing with a pulmonary abscess (see p. 361), it must be remembered that not infrequently, even in severe cases of bronchiectasis, the pleural layers are not adherent over the affected portion of the lung. Accordingly, after resection of ribs, and before opening the

pleura, the visceral and parietal layers must be stitched together, thus minimising the risk of infecting the pleural cavity when the lung is incised.

Experience, however, showed that it is quite rare for a single cavity to be present, and that several bronchi are, as a rule, dilated, so that drainage of one can hardly be expected to effect a cure. Nevertheless, one or two successful cases have been recorded[15] in which complete recovery has resulted, and in others a certain amount of relief—albeit temporary in character—has been given. As a result of the operation the amount of sputum diminishes, the cough becomes less harassing, and fever, if present, tends to subside. Thus, in a case which occurred in the practice of one of us[16]—the first, we believe, of the kind in which the operation was performed—it was very striking to note the extraordinary effect upon the cough and expectoration brought about by draining the cavity. Although the amount of discharge through the drainage-tube did not exceed two ounces, the expectoration, which had previously amounted to sixteen or twenty ounces per diem, was reduced to almost nothing, proving how much of it must have been secreted by the ascending bronchial tract, in response to the irritation produced by the passage over it of the offensive discharge from the dilated portion of the tubes. But the relief obtained is, unhappily, often but temporary; the other dilated and undrained bronchi tend still further to enlarge, the offensive sputum again increases, and the old symptoms recur.

More recently attempts have been made to secure adequate collapse of the lung and diminution in size of the dilated tubes by means of a more or less extensive resection of ribs, while leaving the pleura intact. The soft parts then come to form the covering of the chest wall and the lung falls in. We have seen the operation successful, and the patient's symptoms greatly improved, but the procedure is not free from risk, and we have known a fatal result to follow. Moreover, great deformity of chest results, though this may be to a large extent hidden by a properly adjusted support.

Another operation sometimes performed in these cases, especially when the disease is not limited to the lower lobe, is that of "*rib mobilisation*," as devised by Wilms. This consists in the removal of one to two inches of the posterior por-

tion of the ribs from the first to the ninth or tenth inclusive, and, at a later stage, a similar length of the first five to seven costal cartilages anteriorly. To diminish shock, an interval of a few weeks should be allowed between the operations. As a result the side of the chest falls in, the dilated bronchi collapse, and the symptoms, especially the cough and offensive sputum, are greatly ameliorated. A successful case of this kind was shown at the Clinical Section of the Royal Society of Medicine in 1915 by Mr. Morriston Davies.[17] But here again the procedure is not free from risk. Neither form of operation should, therefore, be undertaken without the most careful consideration, but if the patient's life is rendered burdensome to him by the quantity of foul expectoration brought up by incessant attacks of coughing, then an operation on the lines indicated should be seriously entertained.

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[5] *Report on the Work of the Pathological Department of the Brompton Hospital during the Three Years, April,* 1900, *to April,* 1903, by P. Horton-Smith (Hartley), M.D., Table viii., p. 33. London, 1903.

[6] (*a*) *The Diseases of the Lungs,* by J. Kingston Fowler, M.D., F.R.C.P., and Rickman John Godlee, M.S., F.R.C.S., p. 126. London, 1898.
(*b*) *Loc. cit.,* p. 139.

[7] "A Pedunculated Intrabronchial Tumour (Sarcoma) causing Bronchiectasis," by J. A. Braxton Hicks, M.D., *Proceedings of the Royal Society of Medicine* (*Medical Section*), vol. vii., pt. 2, p. 189, 1914.

[8] "Acute Bronchiectasis," by Seymour J. Sharkey, M.D., *St. Thomas's Hospital Reports,* New Series, 1892, vol. xxii., p. 33.

[9] *Lectures on Clinical Medicine,* by A. Trousseau, New Sydenham Society edition, vol. iii., p. 124. London, 1870.

[10] *Auscultation and Percussion,* by Samuel Gee, M.D., sixth edition, p. 105. London, 1908.

[11] "Remarks on the Treatment of Fœtid Expectoration by the Vapour of Coal-Tar Creasote," by Arnold Chaplin, M.D., *British Medical Journal,* 1895, vol. i., p. 1371.

[12] *Nervous Affections of the Hand, and Other Clinical Studies,* by George Vivian Poore, M.D., p. 276. London, 1897.

[13] "On the Treatment of Bronchiectasis," by T. Grainger Stewart, M.D., *British Medical Journal,* 1893, vol. i., p. 1147.

[14] (1) "The Treatment of Respiratory Affections by Means of Large Medicinal Injections through the Larynx," by Colin Campbell, M.R.C.S., *Transactions of the Royal Medical and Chirurgical Society,* 1895, vol. lxxviii., p. 39. For a discussion upon the paper, see *British Medical Journal,* 1894, vol. ii., p. 1238.

(2) "The Treatment of Phthisis by Intratracheal Injection of Large Quantities of Izal," by Colin Campbell, M.R.C.S., *Transactions of the British Congress on Tuberculosis,* vol. iii., p. 406. London, 1902.

[15] (1) "Two Cases of Bronchiectasis treated by Paracentesis, with Remarks on the Mode of Operation," by C. Theodore Williams, M.D., and Rickman J. Godlee, M.S., F.R.C.S., *Transactions of the Royal Medical and Chirurgical Society,* 1886, vol. lxix., p. 317.

(2) *The Diseases of the Lungs,* by James Kingston Fowler, M.D., F.R.C.P., and Rickman John Godlee, M.S., F.R.C.S., p. 422. London, 1898.

[16] "On a Case of Basic Cavity of the Lung Treated by Paracentesis," by R. Douglas Powell, M.D., and R. W. Lyell, F.R.C.S., *Transactions of the Royal Medical and Chirurgical Society,* 1880, vol. lxiii., p. 333.

[17] (*a*) "Bronchiectasis treated by Rib Mobilisation (Wilms)," by H. Morriston Davies, M.C., *Proceedings of the Royal Society of Medicine* (*Clinical Section*), vol. viii., p. 33, 1915. See also—

(*b*) "Surgery of the Lung and Pleura," by H. Morriston Davies, M.A., M.D., M.C., F.R.C.S., p. 208. London, 1919.

CHAPTER XIV

ON FOREIGN BODIES IN THE AIR-PASSAGES, AND ESPECIALLY IN THE BRONCHI

ALTHOUGH nothing of a definite kind appears to have been written upon the subject of foreign bodies in the air-passages at an earlier period than the end of the sixteenth century, there are indications in literature that the occurrence was not unknown to ancient observers. And doubtless particles of food have "gone the wrong way" occasionally, since the earliest times, from careless eating, under conditions of mental excitement, or in paralysis of the larynx. The ordeal by eating of consecrated bread, which obtained in this country in the early Middle Ages, and the allied mystical trials prevalent in Eastern countries many centuries before, probably had their real basis in that dryness of mouth and irregular action of the muscles of deglutition and respiration produced by excitement, which favour the occurrence of choking attacks from intrusion of food into the larynx. The death of Earl Godwin has in fiction been attributed to this cause.[1]

More strictly within the cognisance of medical history, observations of this accident were recorded by writers at the end of the sixteenth and in the seventeenth centuries, and in 1644 tracheotomy was first definitely advocated[2a] for the removal of foreign bodies from the air-passages, although the operation appears not to have been actually performed for this purpose until fifty years later.

The literature of this limited subject is vast, and the cases quoted are innumerable, involving much repetition. No such admirable exposition of the main symptoms and signs of tracheo-bronchial obstruction has appeared since that by Stokes[3] in his work on *Diseases of the Chest*, published in 1837.

Ætiology.—The circumstances under which foreign bodies obtain access to the larynx are simple enough. Such accidents are common in children with their ancestral tendency to use the mouth as a prehensile organ, and are met with even in adults in eating. Thus, a boy playing with a pea-shooter inhales one of the peas into his larynx; a farm-hand, holding a stalk of barley or bearded wheat between his teeth, has his attention distracted, and the object passing into his mouth is propelled backwards to the fauces by the action of the tongue upon its salient bristles and becomes inhaled into the larynx; a workman, whilst eating rabbit-pie or Irish stew, talks or laughs, and a fragment of bone readily slips "the wrong way." Again, a man smoking a pipe, whilst riding a bicycle or on horseback, may in a sudden fall from an accident, or in an epileptic or syncopal attack, break the stem in his mouth, and inhale it into his larynx. Such are some of the many incidents that have actually resulted in the intrusion of a foreign body into the larynx. Such accidents occur through mere carelessness or frolic, or under any conditions leading to sudden inspiration whilst the mouth is occupied and the patient off guard, as in laughing, coughing, receiving a sudden blow on the back, and the like. They may occur also through impaired sensibility of the protecting laryngeal aperture, be it during the temporary insensibility of deep sleep, anæsthesia, or epileptic, apoplectic or syncopal attacks. Dental, pharyngeal and nasal operations under anæsthesia are not uncommon sources of this accident—a tooth, a septic portion of membrane or growth, or a pledget of lint, besides blood and other fluids, thus obtaining entry to the larynx.

In cases of ulcerative destruction of the epiglottis or larynx, foreign material, and especially fluids, readily obtain access to the trachea; and a similar accident may also occur in paralysis of the glottic appendages, as from diphtheria, bulbar paralysis, and advanced cerebro-spinal lesions.

Liquids in the form of drinks, corrosive fluids, pus, blood, tuberculous matters or portions of growth, may thus penetrate through the larynx to the bronchi and lungs. Corrosive fluids, however, rarely penetrate beyond the larynx, where they set up at first acute spasm and then inflammatory œdema,

which speedily produces suffocation, unless relieved by tracheotomy. A septic broncho-pneumonia or bronchitis not infrequently results from the inhalation of fluids, be they gastric, sanguineous, or purulent.

Except in an academic sense, and for the purpose of complete classification, the occurrences attendant upon the reception of liquids into the larynx are scarcely within the scope of our subject, and may therefore be dismissed without further comment. We may dismiss, too, with a bare mention, those uncommon cases in which matters foreign to the air-passages are introduced into the tubes from below the glottis by ulceration from neighbouring parts; for example, the rupture of suppurating tuberculous bronchial glands into the upper air-passages, leading to the intrusion into them of caseous or cretaceous tuberculous masses. The rupture of abscess or hydatid of the lung, pleura, or neighbouring parts, or the bursting of an aneurism into the bronchial tracts, may also be simply mentioned.

Our chief concern is with the intrusion through the glottis of extraneous and more or less solid bodies. We cannot classify these foreign bodies better than after the manner adopted by Dr. Hoffmann,[4a] and in the table on p. 226 we have ventured to summarise under the same headings an analysis of the cases from his tables and from the supplementary one of Dr. Musser, of which sufficient details are given with regard to certain points of special interest. It will be noted that the table does *not* show the result of the modern method of treatment by bronchoscopy, but, on the contrary, the mortality among cases operated upon and not operated upon before its introduction.

A further group might be added to those mentioned, which would include living bodies, such as *Ascaris lumbricoides*, leeches, small fish, etc., which have in rare instances found access to the air-passages.

From a consideration of the data to which we have referred certain interesting conclusions may be drawn:

Age.—The accident is more common in children and young people. Thus, of the 210 cases tabulated, 140 occurred under twenty years of age, of which 68 were under five years.

Sex.—The male sex predominates. Of 177 cases in which the sex is mentioned, 118 were males and 59 females.

FOREIGN BODIES IN AIR-PASSAGES.

ANALYSIS OF 210 CASES IN WHICH DETAILS ARE GIVEN (ABSTRACTED FROM HOFFMANN AND MUSSER'S TABLES IN NOTHNAGEL'S *ENCYCLOPEDIA*) SHOWING THE AGE, INCIDENCE, SEX, MORTALITY, AND RESULTS OF TREATMENT BEFORE THE INTRODUCTION OF BRONCHOSCOPY.

	Number of Cases.	Ages.			Sex.			Bronchus.			Trachea.	Larynx.	Cases Operated Upon.[1]					Cases not Operated Upon.				Total results Operated and not Operated.		
		Under 5 Years.	Under 20 Years.	Over 20 Years.	Male.	Female.	Sex not stated.	Right.	Left.	Side not stated.			Total Number.	Successful.	Unsuccessful.	Died.	Percentage Mortality.	Total Number.	Recovered.	Died.	Percentage Mortality.	Recovered.	Died.	Total Percentage Mortality.
Class I.: Smooth round pieces of metal or glass (coins, bullets, etc.) ...	20	5	7	8	16	1	3	5	8	6	1	—	10	6	4	—	—	10	7	3	30·0	17	3	15·0
Class II.: Hard, irregular, sharp, and pointed objects (needles, bones, splinters, etc.) ...	106	25	32	49	59	35	12	38	29	20	13	6	39	29	2	8	20·5	67	42	25	37·3	73	33	31·0
Class III.: Soft, rough bodies (heads of grain, etc.)	15[3]	6	6	3	10	4	1	2	3	10	—	—	—	—	—	—	—	15	11	4	26·6	11	4	26·6
Class IV.: Bodies that swell in water (fruit-seeds, beans, etc.)	49	25	19	5	21	13	15	21	13	7	7	1	24	18	1	5	20·8	25	12	13	52·0	31	18	36·7
Class V.: Hard fruit-seeds that do not swell in water (plum-stones, etc) ...	20	7	8	5	12	6	2	4	7	9	—	—	9	3	1	5	55·5	11	8	3	27·2	12	8	40·0
Total ...	210	68	72	70	118	59	33	70	60	52	21[2]	7[2]	82	56	8	18	22·0	128	80	48	37·5	144	66	31·4

[1] By operation is indicated operative extraction with or without tracheotomy.
[2] All the laryngeal cases recovered. One tracheal case died.
[3] In six cases the foreign body discharged through an external abscess.

Position of the Foreign Body.—Of the 210 cases tabulated, the foreign body occupied the trachea in 21, and the larynx in 7. In the remaining 182 it had passed beyond the trachea into one of the bronchi. The substance may be impacted in the glottis, or in the ventricles of the larynx, or in the subglottic space, and in some cases in which it has passed downwards into the bronchus it has been projected from below again into the glottis. Having reached the trachea, the body may be fixed or movable there, and in the latter case a particular sign audible on auscultating the larynx and trachea—the *bruit de grelottement ou de soupape*—has been described as produced by the friction of the foreign body against the glottis and sides of the trachea.[2b] It is a vibratile sound, comparable in some cases to that produced by the sudden application of the plug (*soupape*) to the upper orifice of a water-pipe.

Of the 130 cases in the above table in which definite information is given, in 70 the body entered the right bronchus, in 60 the left. And this is, perhaps, not far from the true proportion, although it has generally been believed, for the reasons first given by Stokes, that the predominance in favour of the right bronchus was greater, the chief reasons being the somewhat wider lumen of the right bronchus, its more vertical direction, and the greater aspiratory power attributed to the right lung. Stokes further pointed out that "the projection or septum dividing the right and left bronchi is not in the mesial line, but decidedly to the left of it, so that a body passing through the glottis will be thus directed into the right bronchus."[3]

Pathology.—The changes *in situ* wrought by the foreign body vary with its shape and texture. If soft and smooth, it may produce no immediate consequence; if hard, rough or pointed, such as fragments of bone, glass or metal, a certain degree of scarification of the membrane is caused, and often immediate capillary hæmorrhage or contusion, with speedy ulceration, will ensue. Again, the condition of the body, whether clean, foul, or septic, will much influence its irritant effects.

Bodies that remain within the precincts of the larynx or trachea cause so much dyspnœa and spasm of the glottis that their speedy removal or the death of the patient generally prevents further local pathological change. Where they

penetrate into the comparatively insentient lumen of the bronchus, they may for long remain undisturbed. Under these circumstances, it is not common for the lumen of the tube to be completely occupied; the body generally becomes attached to one side, and by its pressure and irritation sets up some superficial ulceration, in the depression of which, covered by tenacious mucus, it nestles.

The further and most important changes brought about by the presence of the foreign body occur, first, in the lung territory of distribution of the affected bronchus; secondly, in the pleura and in other parts of the same and the opposite lung; and, thirdly, through septic poisoning and embolic infarction in other parts and organs of the body. These later effects of impaction, which, however, commence very soon, are the result of the partial (or complete) obstruction of the bronchus, retention of bronchial secretions, and their contamination by septic organisms. In this way bronchitis, peribronchitis, bronchiectasis, fibrous inflammation of the lung, and other consequences, ensue.

The characteristic sequel to an obstructed bronchus is bronchiectasis, but there is this peculiarity in the change following upon the reception of a foreign body, that the bronchiectasis so originating at once becomes fœtid. This is not the case with a bronchiectasis that results from partial or complete occlusion by a living tissue, as from the pressure of an aneurism or the intrusion of a growth, provided, of course, that no ulceration or sloughing at the constricted part follows upon the local pressure. The fact is that a foreign body is either septic from the first or very soon becomes so. It then constitutes a focus of septic irritation; the mucous secretions that are poured out become contaminated and purulent, and the contamination spreads downwards through the bronchial tract. Further, owing to the obstruction to the lumen of the bronchus, although it be but partial, the bronchial secretions beyond can never be completely expelled, and a fermenting residue remains. The result is analogous to that which occurs in the urinary bladder when the outflow is partially obstructed. A septic bronchitis is thus produced, with hyperplasia of the submucosa and peribronchitis; the tubes completely lose their tonicity, and passive dilatation follows. The trouble then extends to the lung, to its alveoli and connec-

tive tissue, spreading from the bronchial sheaths. Chronic inflammation and pulmonary fibrosis result, causing a shrinking of the lung texture, and a further widening through traction of the tubes. The rending effects of coughing may have something to do with the dilatation of the tubes in those cases in which the obstruction is incomplete. In some cases an acute pneumonia may supervene at an early date, but this is rare, except when tracheotomy and exploration of the bronchus have been practised.

The subsequent phenomena, such as pleurisy, broncho-pneumonia, empyema and pulmonary abscess, which frequently supervene, are of septic origin. In cases which run their course unrelieved, it is common for one or more of the bronchial dilatations to ulcerate, producing pulmonary cavitation; and in some instances a fistulous tract will be found to extend from one of such cavities to the surface of the lung, leading to empyema or possibly to pyopneumothorax, or, if the pleura be adherent, penetrating to the costal parietes and there producing an abscess. Such a fistula is pathologically identical with those which in other regions lead from bony or other necrotic sequestra.

Lastly, the superior portions of the same lung, and later the opposite lung, become involved in a greater or less degree of bronchiectasis, and in broncho-pneumonic changes of a septic origin, due on the one hand to extension of the morbid processes, and on the other to inhalation of morbid products. Clots from thrombosed and contaminated veins may become detached and conveyed to distant parts as septic emboli, and abscess of the brain from this cause is a not very rare sequel.

Symptoms.—The relation of a case, which occurred before the introduction of bronchoscopy, and which, from the difficulty, if not impracticability, of treating it, ran its course to the end, will perhaps be the most interesting way of bringing together the main symptomatic features of this grave malady.

Mr. T. S., aged fifty-one, a dark, pale man of slender build, in practice as a dentist, had been under observation in 1900 for an attack of renal colic which had not recurred. At 11.30 p.m. on November 29, 1901, a cold, foggy night, he was eating some Irish stew at his home in Bromley, when he felt a piece of bone go "the wrong way." He was seized with severe choking dyspnœa, and at once rushed out, without extra wrap, to a neighbour, Dr. Ilott, who

found him livid and aphonic. An examination of the pharynx with eye and finger revealed nothing. A probang was passed down the œsophagus, after which he said he felt relieved and went home, and Dr. Ilott did not see him again for some months.

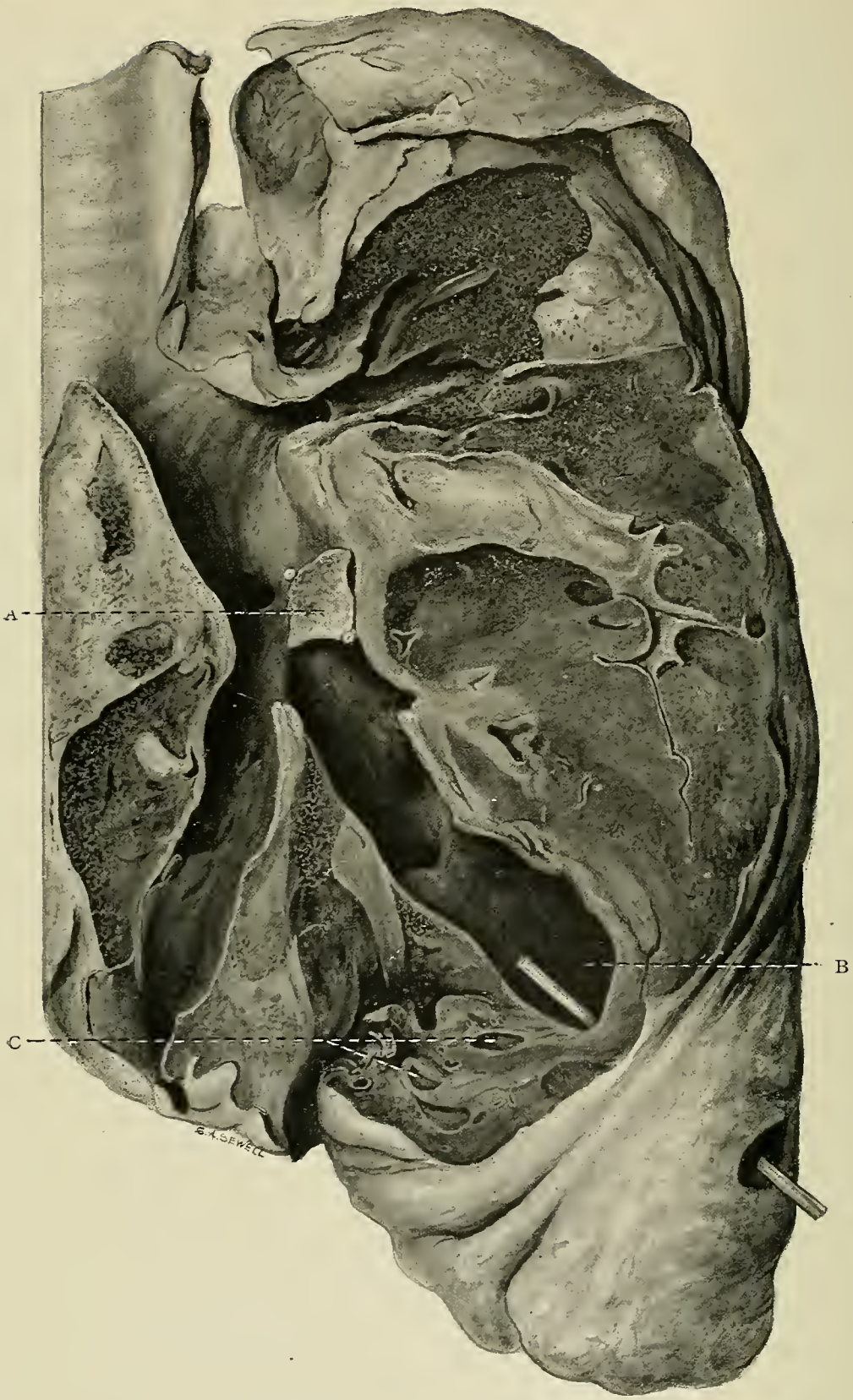
When seen by Sir R. Douglas Powell within a few weeks of this occurrence, he complained of a cough, not attended with any special characters, which he dated from his accident. No physical signs could be detected, except some relative feebleness of respiratory sounds over the right scapular region, and he was himself not clear that the bone had gone into his trachea, or whether his cough was not attributable to a chill caught on the cold night of his attack whilst waiting at the doctor's door.

He went out soon after this to his practice in the South of France, but the cough continued, and within six or eight weeks of his first seizure the expectoration had become copious, purulent, and offensive. He passed through the winter, however, fairly well, and had not lost materially in flesh or strength by the autumn of 1902. Dr. Ilott had seen him in August, and his chief complaint then and afterwards was of purulent and very fœtid expectoration, which came on, and, as he expressed it, "welled up" into his mouth whenever he stooped, as, for instance, in lacing his boots. The chest was everywhere resonant, and on auscultation no morbid signs were detected, except deficient respiratory murmur over the lower part of the right lung. The note of him on November 4, 1902, was that there was a small area of dulness in the right interscapular region, involving the base of the scapula opposite the spinous process; that the respiratory murmur here was weak and semi-bronchial, with a few fine râles; and that over the right lung below the sounds were deficient and attended with slight inconstant wheeze.

The probability of there being a piece of bone impacted in the bronchus was fully recognised, there being still a doubt, however, whether it might not have made an abscess diverticulum from the œsophagus. The patient's own sense of an obscure discomfort led him more than once to express a doubt whether the bone might not have lodged in some point of his œsophageal tract. Dr. Ilott sent him to Dr. Walsham, who took two skiagrams, and recognised a shadow on the right side of the cardiac shadow. Dr. Walsham was kind enough to send the prints, which were excellent, but did not, in our opinion, satisfactorily locate any foreign body. The late Sir James Mackenzie Davidson also was so good as to devote much time and care to the case, with an equally negative result.

Mr. S. continued at his work, and did not complain of any paroxysms of cough or dyspnœa, but only of the annoyance of the expectoration and fœtor of breath. The expectoration was examined on several occasions in the autumn of 1902. It consisted of almost pure, thin, diffluent pus, foul-smelling, and containing very little mucus. Numerous micrococci were present, but no tubercle bacilli nor elastic tissue elements. At this time he was seen in consultation with

PLATE XIII



FOREIGN BODY IN BRONCHUS.

To face p. 231.

FOREIGN BODY (PIECE OF BONE) IN BRONCHUS, LEADING TO CYLINDRICAL BRONCHIECTASIS, SEPTIC BRONCHO-PNEUMONIA, AND DEATH FROM PNEUMOTHORAX

At A a piece of bone is seen embedded in the main bronchus to the lower lobe of the right lung. Below this the bronchi are dilated in a cylindrical manner, and at B the dilatation expands into a cavity the size of a walnut. Adjoining this, septic broncho-pneumonia had occurred, leading to pneumothorax, and a glass rod is seen passing from the dilated bronchus through the softened lung and through a perforation in the pleura the size of a sixpence. At C other smaller bronchi are seen dilated and cut across.

From a gentleman, Mr. T. S., aged fifty-one, whose case is described in the text, with the result of the autopsy (see p. 229).

(From the Brompton Hospital Museum. $\frac{4}{5}$ natural size.)

PLATE XIII

Sir Rickman Godlee, but it was decided that no surgical interference could be recommended, as the exact locality of the foreign body, if present, could not be demonstrated.

He went out to the South again after Christmas, and returned with more evident signs of advanced disease upon him. His finger-ends were enlarged, he had lost flesh considerably, and in the course of the summer he had had several attacks of rather severe hæmoptysis. In June, 1903, his temperature was raised, his breathing quicker, and the expectoration copious, fœtid, and frequently blood-stained. There was impaired resonance at the right base, and over an area in the mid-scapular line in the region of the ninth and tenth ribs the respiration was distinctly cavernous at times, with râles of a liquid or sucking character. Some pleuritic friction was also audible, and a few crepitations were heard at the left base. The question of surgical interference was again considered with Sir Rickman Godlee, and Sir James Mackenzie Davidson took another skiagram. On this occasion a distinct square shadow could be seen corresponding with the area over which cavernous breathing was audible, and which moved up and down with deep respiratory movements, but this was not thought to be due to the presence of a foreign body in that position.

About July 30 he had a severe hæmorrhage, for which Dr. Ilott treated him, and on its subsidence, at the request of Dr. Ilott, his admission was secured into the Brompton Hospital. He became rapidly worse, however, and on August 10 was seized with acute pain in the side, and was found by Dr. Kidd to have developed pneumothorax, followed by rapid effusion. Sixteen ounces of thin, purulent fluid and some air were drawn off to relieve distress. He died on August 14.

Post-mortem Examination by Dr. H-S. Hartley.—The right lung was collapsed, except for adhesions at the apex and in the region of the diaphragm. The pleural cavity contained air and about one pint of thin pus. An opening in the pleura, the size of a sixpenny-piece, was found near the base of the lower lobe in the posterior axillary line (Plate XIII). The patch of lung here presenting was softened, and in it a hole was found, which admitted a large probe, and which communicated with the main bronchus to the lower lobe. In this bronchus, about an inch from its commencement, a triangular piece of bone, $\frac{3}{4}$ by $\frac{5}{8}$ inch, was found; it lay in an ulcerated patch, and could be removed with ease. The bronchi below were cylindrically dilated and much congested, with foul-smelling contents, the chief bronchus expanding near its extremity into a cavity of about the size of a walnut, which corresponded in situation to the point over which cavernous respiration was heard during life. From the lower end of this cavity there extended a sinus downwards and outwards to an area of broncho-pneumonic softening at the surface of the lung, where the perforation had occurred. The lung tissue of the right lower lobe had undergone fibrosis. The bronchi in the upper and middle lobes

above the piece of bone were distinctly dilated, but less so than in the lower lobe.

In the left lung the bronchi to the lower lobe were dilated and inflamed, and those in the upper lobe were a little larger than natural. This (left) lung showed but little fibrosis, but presented patches of septic broncho-pneumonia.

This case, when we look back upon it from the end to the beginning, illustrates most of the features characteristic of the presence of a foreign body in the bronchus, and in the course of comments upon each set of symptoms as they presented themselves we shall be enabled to bring before the reader the main features of the disease.

We have (1) a definite choking attack. (2) A lull into comparative comfort, with a vague sense of general uneasiness or irritation. (3) A cough then comes on, which may or may not—but in this case did not, at all events, in the earlier periods—present special features. (4) The expectoration becomes more copious and offensive. (5) The physical signs, at first very obscure, gradually assume the characters of chronic basic one-sided bronchiectasis, with fibrosis of the lung. (6) Hæmoptysis occurs towards the end of the illness. (7) Some acute incidents, usually of septic origin, such as broncho-pneumonia, abscess, empyema, in this case pneumothorax, bring the sufferings of the patient to an end. (8) The X-rays may or may not, according to its structure, reveal the presence of the foreign body. In this case they did not do so.

A stormy onset, an acute choking fit, is the rule in cases of foreign bodies entering the trachea, but the intensity of the attack varies much with the character of the body inhaled and the state of the patient. It is obvious that a rough, angular and hard substance will excite much more irritation than one which is smooth, rounded, or soft. In some cases the body is inhaled when the patient is asleep, or under an anæsthetic, or in a fit, and in this way may excite no preliminary spasm. Thus, a molar tooth was found post-mortem by our colleague Dr. Young in the bronchus of a patient who had died with uræmic convulsions in the Middlesex Hospital. It had probably become dislodged in the nurse's endeavour to prevent the clenching of the jaws in the convulsions during which the patient died; it had caused no symptoms, and had evidently only been in the bronchus a short time before death. A piece

of tooth-filling or an extracted tooth may similarly be inhaled into the bronchus during a dental operation under an anæsthetic.

A few years ago one of us was consulted in the case of a gentleman with severe incessant and paroxysmal cough, attended with the expectoration of a glairy, blood-stained mucus. He had been riding in the Park, and was supposed to have had a fit, causing him to fall off his horse. He was carried home insensible, and on return of consciousness presented the symptoms described. There were crepitations, which were regarded as due to blood within the tubes at the left subclavicular region, with very feeble breath-sound. The case had not the characters of tuberculous disease, but no history of foreign body could at the time be elicited, although carefully inquired for. He was not seen again by us after the first few days of his illness until some three months later, when, having suffered meanwhile from a severe cough and increasingly purulent expectoration, he had expelled with a severe paroxysm a pipe mouthpiece about ¾ inch long. He now recalled to mind that he was smoking a pipe at the time of his accident!

The practical point, then, in all cases is never to pass over without most careful scrutiny any history of attack suggestive of the inhalation of a foreign body, or any occurrence preceding the development of symptoms that may suggest the possibility of such reception.

The lull of symptoms that often follows the initial dyspnœa for some days, and even some weeks, and which is due to the penetration of the foreign body beyond the sensitive territory of the glottis into the comparatively insensitive bronchus, tends to put the doctor and the patient off guard as to the true nature of the cause. In the case of Mr. S., he himself and some who saw him in the earlier stages were, for this reason, sceptical as to the foreign body having entered his larynx, and were inclined to attribute his cough to a bronchial cold caught from exposure on that November night. A diagnosis, therefore, of the presence of a foreign body having been made, it is not to be put aside by, nor is safety to be augured from, a temporary absence of further symptoms. Many authors since the time of Louis have borne witness to the intervening periods of calm that often occur between the first

attack and the occurrence of secondary symptoms. Dr. Hoffmann[4b] observes: "In many cases, after an initial violent fit of coughing, weeks and months may elapse during which the patient's comfort is not disturbed."

There was not in this case anything distinctive about the cough, but in many others, such as that of the gentleman alluded to who inhaled the pipe mouthpiece, the cough is very paroxysmal, and has even been mistaken for whooping-cough.[5] Sometimes, as in the case of a child recorded by Mr. Kellock,[6] the patient, after the first severe paroxysm, will have intervals of comfort for some hours, but will wake up in the night, or be seized after crying with intense dyspnœa and lividity. It is evident that in these cases the foreign body becomes projected from its nidus in the relatively insensitive bronchus towards the larynx, which is supplied by the sensitive superior laryngeal nerves.

As the case goes on, if the foreign body remains, the cough continues, and is accompanied by expectoration, at first mucous, but soon becoming purulent, and then fœtid, and more and more copious in amount. A symptom which was so pronounced in the case which we have described, namely, the welling up into the throat of fœtid purulent matter on stooping, is a more or less prominent feature of bronchiectasis, and we presume that it was due in this case to the positional displacement of the foreign body permitting the more or less pent-up secretion from below to pass.

Hæmoptysis is sometimes an early symptom. More frequently it occurs in the later stage, and it is naturally most common in those cases in which hard, irregular bodies have been inhaled.

The physical signs are as a rule slight to the point of obscurity. Should the body be movable in the trachea, the *bruit de grelottement*, already described, may be audible, but otherwise feebleness of breath-sound over one lung below the seat of the obstruction is the only constant early sign. There may soon be developed, as in the case of Mr. S., at and about the point of obstruction, an area of impaired percussion note, with fine râles, due to hyperæmia in the neighbourhood of the offending body. Some variable sibilant or mucous sounds are scattered over the affected side. If the obstruction be at all complete, the movement on that side will

be obviously lessened. Later some dulness will be found at the base of the lung; perhaps some friction may be heard, or the signs of effusion may gather there. As a rule, as the illness proceeds, the signs become those of a gradual condensation of the lung, varying from time to time from mere silence to patchy bronchial or even cavernous breath-sound and localised pectoriloquy, always, however, on the scale of weakened breath-sound, and indicative of a fibrotic and bronchiectatic lung. An empyema may form, or an abscess may develope in the lung, or a fistula may extend from the obstructed bronchus through the chest wall, causing a thoracic abscess or a purulent pneumothorax, as happened in the above related case. At first the signs are always one-sided; later on, towards the end, septic incidents occur in the other lung or in distant parts, cerebral abscess being not infrequently observed.

Diagnosis.—The diagnosis rests upon an accurate history of seizure, followed by the symptoms and signs of an incomplete obstruction to the function of one lung, with bronchial irritation, and subsequently bronchiectasis and fibrosis.

It will often happen that the patient is only brought to us in the later stage of fibrosis and bronchiectasis, and then the difficulties may be great in distinguishing the case from one of abscess of the lung, empyema, or phthisis. An examination into all its features will, however, usually lead us to recognise that we have to deal with a one-sided bronchiectasis, and we must from that proceed carefully to inquire into the history of attack, and for any circumstances that may point to the reception of a foreign body. It must be remembered that a large proportion of cases of bronchiectasis are one-sided; but when a one-sided bronchiectasis comes on apparently as a primary disease, and cannot be explained by a history of an antecedent bronchitis, pneumonia, or pleurisy, the possible presence of a foreign body should be carefully inquired for. It is, again, to be remembered that the second lung becomes involved in the later stages of all cases, whatever the source of the bronchiectasis may be.

An examination by the X-rays should be made, but in diagnosis too much must not be expected from this source. When the foreign body is metallic or of pebbly character, as in Class I. in our table, in some cases of Class II., and perhaps

in Class V., it will be well shown up and located in a skiagram. But when the obstruction is of cartilaginous or fleshy material, or if, being of bony structure, it be impacted beneath the shelter of the scapula, it may be impossible to detect it by this means. Looking back upon the case described, due weight was perhaps not attached to the difficulty of demonstrating the bone in this position.

At the time at which Mr. S. was under observation the method of bronchoscopy or the direct examination of the interior of the trachea and bronchi, first brought into practical use by Killian, was in its infancy. It is now familiar to all physicians interested in laryngological and chest work, and it is in cases like the present that it is of especial value. Had it been available, the piece of bone would, in all probability, have been detected, and, judging from the loose manner in which it lay in the bronchus at the autopsy, its removal by a little careful manipulation would have been possible. The method should be employed in all cases in which doubt remains as to the presence of a foreign body. Its value in treatment we shall consider later.

Prognosis.—The reception of a foreign body into the air-passages is a serious occurrence, the gravity of which varies with the nature of the material inhaled. Excluding from our reckoning such trivial matters as small breadcrumbs, flies, etc., that may pass into the trachea and occasion only temporary inconvenience, a glance at the table on p. 226 shows that the total mortality among 128 cases not operated upon was 37·5 per cent., reduced to 31·4 per cent. if we include those treated by operation, and that this mortality corresponds closely with that of the 106 cases in Class II. of hard, irregular bodies. This latter class includes the greatest number, and is, therefore, perhaps the most trustworthy for percentages. The bodies included in it (teeth, bones, pipe-stems, needles, peg-top points, etc.) are highly irritating, and often directly septic. In Classes IV. and V. the foreign bodies are of a kind likely to become, either by their primary form (plum-stones, etc.) or their secondary swelling when soaked (wheat, maize, beans, etc.), more tightly jammed into the bronchial calibre. The least harmful bodies seem to be the smooth impermeable bodies, such as coins, bullets, buttons, beads, etc., giving a mortality of 15 per cent.; but the number

of these cases is comparatively small from which to draw percentage conclusions.

In regard to the influence of the older methods of operative treatment upon the mortality, we may note that of 82 cases in which operative treatment—mostly tracheotomy—was adopted, 22 per cent. died; whereas, of 128 cases in which no operation was attempted, 37·5 per cent. died.

Treatment.—Two principles are laid down by Sir F. Semon and Dr. Watson Williams[7] in their article upon the presence of foreign bodies in the air and upper food passages: first, no foreign body the presence of which has actually been detected should be permitted to remain impacted, without an attempt to remove it by every justifiable means, even although at the time it may not produce active symptoms; secondly, no attempt should ever be made forcibly to ram down an angular or pointed foreign body. It is unnecessary after what has already been said to dwell upon these rules of practice. When the foreign body has penetrated to the bronchi, the first dictum still holds good.

These being the fundamental ideals with regard to treatment, we may describe the main methods of procedure as follows:

If the first paroxysm has passed, and the services of an operator skilled in bronchoscopy are available, a search should be made without delay by this method for the foreign body, and its removal attempted. With the recently improved manipulative methods and instruments designed by Killian, Brünings, Von Schrötter, and others, the facilities for extraction have been greatly increased, and the attempt is now successful in the majority of cases. The pipe-stem referred to in the above-quoted case, although ultimately expelled by Nature's efforts, might readily have been detected by bronchoscopy, and would probably have been easily removed by this means.

Whether *superior* or *inferior bronchoscopy* should be performed—the former being the introduction of the tube through the mouth and glottis, and thus into the trachea and bronchi; the latter its introduction through a previously performed tracheotomy wound—must depend upon various considerations, the most important of which are the size and character of the foreign body, but we may add that in the

majority of cases superior (or per-oral) bronchoscopy will prove successful.

Professor von Eicken,[8] of Freiburg, reported in 1909 upon 303 patients of all ages (195 children under fifteen and 108 adults) treated by superior or inferior bronchoscopy. Of this number, in 233, or 76·9 per cent., the foreign body was located and extracted, and recovery ensued. In 36 (11·9 per cent.) the result was only partially successful, the lung being in many cases already so diseased that, in spite of the successful removal of the foreign body, death could not be averted, and at most only partial improvement followed. In 34 (11·2 per cent.) the operation was unsuccessful, the foreign body in several cases not being found, or its removal proving impracticable. Included in this group are also four patients who succumbed to cocaine-poisoning, and one who died under chloroform. Two children also died of suffocation from the fracture of the foreign body (a softened bean) during an attempt at its removal by superior bronchoscopy, and the consequent inhalation of the fragments.

Since 1909 the results of this method of treatment have improved with advancing experience, and at the 17th International Congress of Medicine held in London in 1913, Dr. Chevalier Jackson,[9] who introduced the discussion on this subject, reported that in the last 182 consecutive cases of bronchoscopy for foreign bodies which he had had under his care, in 177 the foreign body was successfully removed. In 3 only had the patient died—a mortality of 1·7 per cent. Dr. Jackson uses only per-oral bronchoscopy, being unconvinced of the advantages attributed to tracheotomic bronchoscopy, while the disadvantages are many and obvious.

Without pressing the actual figures too far, we may note that these results contrast most favourably with those obtained by older methods of treatment, as recorded by the late Mr. Durham,[10] Weist,[11] Preobraschensky,[12] and others, and as indicated on our table on p. 226, and there can be no question that direct examination and extraction is the right procedure to adopt, provided the surrounding circumstances permit. When, however, the requisite instruments and skill are not available, and if the first paroxysm has passed, and if we know also that the foreign body is of the smooth, hard variety, referred to in Class I. of our table, simple inversion,

or inversion with tracheotomy, may be tried. Under this treatment the patient is inverted with slapping or thumping shocks between the shoulders. This should only be done with instruments ready at hand for tracheotomy should the paroxysm of dyspnœa consequent on displacement of the body to the glottis be too severe.

In a considerable number of cases the foreign body has been thus expelled. A famous instance of the kind, in which, however, tracheotomy was performed, is that recorded by Sir Benjamin Brodie[13] of the celebrated engineer Brunel, who, whilst playing with some children, inhaled a half-sovereign into his bronchus. On the sixteenth day, the patient having been placed prone, with his head and shoulders slanting downwards, "he immediately had a distinct perception of a loose body slipping forward along the trachea. A violent, convulsive cough ensued. On resuming the erect position, he again had the sensation of a loose body moving in the trachea . . . towards the chest." Two days later, in presence of Dr. Chambers and some other colleagues, the experiment was repeated by means of an inclined, hinged platform. On striking the back with the hand over the right bronchus, with the head lowered to an angle of 80 degrees with the floor, violent cough was set up without result, and the experiment was twice repeated, on the third time causing such an alarming degree of choking that it was not further proceeded with. A week later tracheotomy was performed, and forceps introduced in an attempt to seize the coin, but without result. The tracheotomy wound was kept open, and after a further delay of ten days the patient was again inverted, and after two or three strokes on the back the coin was expelled through the mouth. "No spasm took place in the muscles of the glottis, nor was there any of that inconvenience and distress which had caused no small degree of alarm on the former occasion."

In cases of angular bones, pointed instruments, and bodies of like nature, if more modern methods of treatment be not available, tracheotomy should be performed, and an attempt made to extract the body by means of slender forceps, or, perhaps, a loop of silver wire. *Pneumotomy*,[14] or the cutting down through the chest wall directly upon the foreign body, is an operation which, though by no means so hazardous as formerly, is yet not free from risk, and should be reserved for

those rare cases in which other methods have failed. We have, on page 164, related a successful case bearing upon this subject.

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(*b*) Guyon, *loc. cit.,* p. 713.

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[11] "Foreign Bodies in the Air-Passages," by J. R. Weist, M.D., of Richmond, Indiana, *Transactions of the American Surgical Association,* Philadelphia, 1883, vol. i., p. 117.

[12] "Ueber Fremdkörper in den Athmungswegen," von Dr. S. S. Preobraschensky in Moskau, *Wiener Klinik,* 1893, Nos. 8-10.

[13] "An Account of a Case in which a Foreign Body was lodged in the Right Bronchus," by Sir Benjamin C. Brodie, Bart., F.R.S., *Transactions of the Royal Medical and Chirurgical Society,* June, 1843, vol. viii., p. 286.

[14] (*a*) "A Case of Pneumotomy for Foreign Body," by Thomas H. Kellock, M.C., *Proceedings of the Royal Society of Medicine* (*Clinical Section*), vol. vi., p. 64. 1913.

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CHAPTER XV

ASTHMA

On more closesy regardng the several varieties into which asthma is divisible for clinical convenience, it will be observed that they represent merely ætiological subdivisions of a disease which consists essentially of a *paroxysmal dyspnœa from disturbed innervation of the bronchi, leading to spasmodic contraction of these tubes.*

Pathology.—The theory that asthma is thus the result of bronchial spasm, though held by many authorities from Laennec onwards, has not been universally accepted, some believing that the disease is due to a sudden swelling of the bronchial mucous membrane from turgescence of its blood-vessels; others that it results from a special form of inflammation, a "bronchiolitis exudativa" (Curschmann), affecting the smallest tubes. For a consideration of the numerous other theories which have been held from time to time concerning the true nature of the disease, we must refer the reader to Salter's[1a] standard work, in which these theories are well related.

That true spasmodic asthma is a malady in which the nervous system is closely concerned is supported by the following clinical considerations:

1. An attack of asthma may supervene in the course of a few minutes, or even seconds, in a person in whom there can be found no evidence of chest disease.

2. Of the organic lesions which can be said in different cases of asthma to have led up to the spasmodic seizures, there are none which singly or combined are proper to that disease. Asthma, it is true, leads on to certain pathological conditions recognisable during life, notably emphysema, chest deformity, cardiac disease, and visceral congestions; but these lesions are distinct mechanical consequences after long continuance of the disease, and in their earlier stages are totally

inadequate to account for the asthmatic phenomena. We have elsewhere drawn attention to the intimate analogy between pure asthma and vaso-motory angina, especially in these respects.*

3. The family histories and mental proclivities of many asthma patients, the capriciousness and intermittent character of their attacks, recall to mind such affections as neuralgia, migraine, epilepsy, and emphasise the part played by the nervous system in the pathology of the disease.

We may now pass to a consideration of the question whether a spasm of the muscular tissue of the bronchioles, the contractility of which was originally demonstrated by the late Dr. C. J. B. Williams,[3] is the cause of the asthmatic attack. The arguments in support of this theory have received very cogent and, as it appears to us, convincing support, from the work of Professors Brodie and Dixon.[4]

These observers have shown, by means of an ingenious method of recording the volume of air entering and leaving the lung at each respiratory act, that peripheral stimulation of the vagus leads in the corresponding lung to a contraction of the muscular walls of the bronchi and a constriction of their lumen, resulting in a great diminution of the volume of air inhaled. With this change an over-distension of the lung, as in asthma, was frequently observed. There can be no doubt, therefore, that under the influence of the nervous system marked changes of calibre in the bronchioles can be produced, and Dr. Watson Williams[5] is of opinion that the asthmatic paroxysm is due to an exaggeration of the contraction stage during expiration of the normal respiratory rhythmic movements of the bronchi, to which we have referred earlier (see p. 8).

The influence of drugs was next investigated, and Drs. Brodie and Dixon found that the injection of muscarine and pilocarpine led to great constriction of the bronchioles due to peripheral stimulation of the vagal nerve-endings, and that if atropine, hyoscyamine, or hyoscine were then injected, dilatation at once supervened from paralysis of the same nerve-endings. The experiments with muscarine were especially interesting, for the animal under observation suffered

* See Table comparing Asthma with Angina Pectoris Vaso-motoria, in Sir Richard Douglas Powell's article on "Angina Pectoris."[2a]

from a typical asthmatic paroxysm, with dyspnœa, distended chest and scattered sibilant râles, until the injection of atropine cut short the attack. The experiments with chloroform and ether were also instructive, the results showing that these drugs, when absorbed through the bronchial mucous membrane, led to paralysis of the nerve-endings, thus explaining their good effect in the treatment of the disease. Of great importance, too, was the observation that a definite reflex contraction of the bronchioles could be obtained by stimulation of the nasal mucous membrane.

The theory of bronchial spasm would appear, therefore, adequate to explain the asthmatic attack. Drs. Brodie and Dixon draw attention also to certain facts which militate against the theory of vascular turgescence, which has found in the past not a few supporters. They point out, in the first place, that it is illogical to assume that a sudden and marked vascular engorgement may be expected in the bronchi, simply because it occurs from time to time in the nasal mucous membrane, seeing that the structure of the two is quite dissimilar. "The nasal mucous membrane," they remind us, "especially over the inferior turbinate bone and lower nasal passages, is extremely vascular, and in many parts large venous plexuses are found encircled by bundles of muscular fibres, thus forming a sort of cavernous erectile tissue (Klein). The bronchial mucous membrane, on the other hand, is thin, and possesses what is, in comparison, a relatively insignificant blood-supply." Again, in the artificial asthma produced by muscarine, the bronchi were not found engorged, nor was any excessive secretion found in them. Further, the marked turgescence of the bronchial mucous membrane, produced by the experiment of clamping the pulmonary vein, never gave rise to diminution of air entry at all comparable to that seen when the bronchioles were made to contract.

The little pellets of sputum which are expectorated towards the end of an attack of asthma should not be regarded as a sign of bronchitis, but are to be attributed rather to mechanical hyperæmia, induced by disturbed intrathoracic pressure acting for a considerable time upon the obstructed tubes.

Ætiology.—A considerable number of cases of asthma are attributable to inherited predisposition, and in many instances

what is termed a "neurotic family history" obtains, although evidence of direct inheritance of asthma may be wanting. Epilepsy, insanity, neuralgia, hysteria and asthma, are indeed diseases all within the range of interchangeability in families.

Asthma is said to be more common in males than in females, though this does not happen to be in accord with our own experience, and it is stated by Dr. Salter and others to occur most frequently in the first decade of life. It is, however, often first manifested between puberty and early middle life, and in females about the menopause. Cases commencing in middle or later life, and they are not very infrequent, have in our experience mostly supervened upon influenza, others from the gouty plethora induced by undue liberality in diet, and a few—one notable instance—from nervous exhaustion consequent upon the cares and work of official life. In connection with the ætiology of asthma, especially that variety commencing in early life, eczema will occasionally be recorded; in later life sometimes urticaria.

Exciting Causes of Asthma.—In the most characteristic cases of asthma the exciting causes of the attack are operative in a patient with an idiosyncrasy, often hereditary, which is manifested in an undue sensitiveness of his respiratory centres. The exciting causes are of central or reflex origin, and are often toxic in nature. We may first consider toxic causes, since they are often associated with the others.

It has recently been shown by Dr. John Freeman[6] and by Dr. I. Chandler Walker[7] of Boston, and others, that in many cases the asthmatic attack is in reality of anaphylactic origin, the patient being hypersensitive, anaphylactic, to some protein, which may be present in *pollen*, *animal hairs*, *food*, or *bacteria*, and which, when absorbed, so affects him as to precipitate the seizure.

Dr. Walker examined 400 patients suffering from asthma, and found that 191, or 48 per cent., gave a positive skin test, as shown by a marked urticarial wheal produced on scarifying the skin through a solution of the protein in question, thus proving the patient sensitive to the variety of protein tested.

With regard to the class of protein at fault, it has been shown that hypersensitiveness to a *food protein*, whether of cereals (and especially wheat), or egg, or milk, is more frequently to be observed in cases commencing in infancy, 23 of Dr.

Walker's 34 cases being under two years of age. In later years, when caused by food protein, the asthma is more often attributable to other articles of diet, such as fish or potato.

The same relationship to age of onset is true, but in a less degree, in regard to the protein present in hairs (chiefly those of the horse, less commonly the cat), or that occurring in feathers, which appears to be responsible for a good many cases of asthma commencing before the age of ten, and less often as the age of onset advances. Sensitisation to pollen protein, producing so-called "hay asthma," is more common in cases commencing in young subjects, but is by no means limited to such patients; while bacterial toxines, notably of staphylococcal or streptococcal origin, may be associated with asthma occurring at any age.

We may here note, as probably of analogous explanation, the alarming symptoms of dyspnœa, failure of respiration, and collapse, which have sometimes quickly followed the subcutaneous injection of serum, whether anti-diphtheritic or other, when given for therapeutic purposes in asthmatic subjects, who are often anaphylactic to horse serum. Of 28 such cases collected by Dr. Gillette,[8] 15 proved fatal (see p. 264).

Presumably these protein bodies act directly upon the medullary nerve centres, and perhaps asthmatic phenomena sometimes observed in renal disease from retained urinary products may be similarly caused. We have already alluded to the effect of muscarine.

It is only possible—so far as present technique serves—in some 48 per cent. of patients to trace asthmatic attacks to such proteid sensitisation. The tests in the remainder of Dr. Walker's cases proved negative. Other cases we must refer to the more obvious sources of *reflex irritation.* In some cases excitation of some point of the naso-pharyngeal tract may be at the origin of the trouble. The asthma has thus been traced to the presence of polypi, adenoids, or sinus disease, and treatment of the nasal affection has led to a diminution in the number of attacks. We have also met with a few well-marked cases in which the asthma paroxysm has commenced with a turgid state of the turbinate membrane, causing complete occlusion of the nares. In other cases, again, asthmatic attacks arise from more direct bronchial irritation, as in bronchitis, plastic bronchitis, or from inhala-

tion of inorganic dust, or exposure to acrid vapours, or to certain atmospheric influences to which the patient may be peculiarly sensitive.

Amongst other reflex causes are gastro-intestinal disturbances, especially flatulent distension of stomach or bowels, and loaded colon; but it would be difficult to affirm how far the influence of certain proteins may not also be concerned in this causation. Less common sources of reflex irritation are connected with uterine disturbances, whether menstrual or climacteric, and those derived from excitation of cutaneous nerves.

Cutaneous affections, especially of the eczematous type, are not infrequent precursors of asthma in young children, and the late Sir Andrew Clark was of opinion that asthma occasionally originated in an urticarial condition of the air-passages.

The emotional causes which may be responsible for the attack no doubt act in a reflex manner, impulses originating in the cerebrum stimulating the lower centre in the medulla. We have recorded an interesting case in which asthma, vasomotor angina, and menopausal sweatings have shown an interchangeability.[2b]

Clinical Varieties of Asthma.—In accordance with the varying nature of the exciting cause, diverse clinical varieties of the disease have been described, of which the following may be mentioned:

(1) *Idiopathic, Essential, or True Spasmodic Asthma.*—This has been held to include those cases of true neurosis in which no lesion, whether pulmonary or other, can be found, and in which the exciting cause of the attack, if any can be recognised, is some mental or emotional disturbance, for example, mental shock, violent emotion, severe anxiety; or, if material, one of a very slight and ephemeral kind, such as a brilliant light, a transient odour, the momentary application of cold to the surface, and the like. This form of asthma, as already said, can be best compared to neuralgia, epilepsy, migraine, perhaps also mania. The neurosis is inherited either directly or through some of the allied forms mentioned. The attacks are more distinctly periodic than in any of the other forms of asthma, and with the completion of each attack the peculiar neurosis often seems to be for a time worn out.

(2) *Catarrhal or Bronchitic Asthma.*—In this variety catarrhal symptoms precede and attend those characteristic of asthma, the dyspnœic phenomena being due to direct irritation of the bronchial nerves, or more probably, as we have seen, to their reflex excitation. Bronchitic asthma is but a catarrhal affection of the bronchial tubes in a subject predisposed to asthma; the special character of the disease being derived from the individual peculiarity, not from the catarrhal affection which commonly arises in the ordinary way. In this affection during the acute stage the spasm and consequent dyspnœa are more or less continuous. Subsequently, with the greater freedom of secretion and expectoration, the dyspnœa occurs chiefly in the early morning after a few hours' sleep, when the accumulation of mucus in the tubes is greatest, and with its expulsion the spasm ceases.

In cases of old-standing emphysema fresh bronchial catarrh is often attended with dyspnœal seizures of an asthmatic character which are due to obstruction of the tubes by mucus disturbing the, at all times, unstable respiratory equilibrium of the emphysematous subject. Only in popular parlance can such cases be called "asthma."

(3) Of *dust asthma* we shall detail a well-marked example arising from the inhalation of wood dust (see p. 264). Other irritants have similar effects upon a certain proportion of those exposed to them.

(4) *Hay asthma* is a variety of the disease which is often associated with that intense catarrh of the conjunctivæ and of the nasal mucous membrane which is termed "hay fever." The researches carried out at Hamburg, under the direction of Professor Dunbar,[9] and since by many other observers, have greatly extended our knowledge of the ætiology of this complaint, and have shown that, in patients whose nervous system is sufficiently receptive, the pollen of very many grasses, plants, and flowers is capable of producing the attack. Among these we may mention rye, oats, barley, meadow-sweet, golden rod, chrysanthemum, and aster. In England the complaint is chiefly produced by the pollen of hay (*Anthoxanthum odoratum*), in Germany by that of the flowering rye; in either country the early summer months, May to July, are those in which the disease is met with. In America, in addition to the attacks in early summer, a severe

form of the complaint is met with in the autumn, the so-called "autumnal catarrh," which is produced by the pollen of certain grasses which flower at this season of the year.

The pollen of the various plants in question contains, as demonstrated by Professor Dunbar, an active protein, soluble in saline solution, minute doses of which produce in sensitive subjects the symptoms of the malady, and must therefore be regarded as its cause. The pollen floating in the air finds its way into the eye or nose, its protein is extracted and absorbed, and thus gives rise to the lachrymation, sneezing, and running from the nose which form so familiar a picture. In a smaller proportion of cases, and generally some hours later during the night, an attack of asthma follows.

(5) *Nasal asthma*, as the name implies, comprises that group of cases to which we have already alluded, in which the asthma has been traced to a definite source of irritation within the nose. In such cases relief may be given by operative treatment, a question to which we shall recur later.

(6) The term *peptic asthma* includes those cases in which the asthmatic attacks bear a definite relation to gastro-intestinal irritation, flatulent distension of the stomach, and so forth, and it is not to be doubted that many cases of asthma met with are in this sense manifestations of dyspepsia. The class of peptic asthma may, however, with great practical utility be enlarged. Thus, Salter[1b] expressed the belief that the introduction of food frequently gave rise to asthma, not by irritation of the alimentary canal, but by absorption into the veins of materials perfectly normal to the stage of the digestive process, but which in the asthmatic stimulated the unduly sensitive pulmonary nervous system to produce spasm; and in this connection we must recall that certain articles of diet, especially saccharine substances and other carbohydrates, prove unsuitable to some asthmatics. We have already alluded to cases, chiefly of children, in which the patient is anaphylactic to the protein of certain foods. In the gouty subject, the blood circulating through the nervous centres is charged with imperfectly changed and effete material, and attacks of "gouty asthma" may thus originate. It may well be believed, too, that in cases of loaded colon asthma is sometimes set up by the absorption of toxines and putrefactive matters.

(7) *Cardiac asthma* belongs to a different group from that which we have been considering. It is in reality a consequence of heart failure, whether the result of fatty and senile degenerations or of valvular disease with incomplete or damaged compensation. Most commonly the attacks occur during sleep or towards the morning, when the blood-pressure and general vitality are low. Signs of passive congestion of the bases of the lungs, more especially on *one* side, may generally be detected, and some blood-staining of sputa is not infrequently observed to follow the attack.

(8) In *uræmic asthma* œdema of the lungs is generally present. We have seen cases, however, in which the morbid material in the blood seemed to be more directly the excitant, thus bringing them more into line with true asthma.

Symptomatology.—The subjects of asthma present, in the advanced periods of their malady, a characteristic physiognomy. Thin, of nervous temperament, grave-featured, with slightly depressed angles of the mouth, high-shouldered and round-backed, they carry the impress of suffering in feature and build, and one is sometimes surprised at the power of work, keenness of wit, and capacity for enjoyment with which these persons are gifted. It requires many severe attacks, however, to bring about these characteristic appearances, and in the intervals between the earlier seizures there may be nothing in the physiognomy of the patient symptomatic of asthma. In the later periods of the disease, and at an earlier date after a recent attack, there may be quickened breathing, accompanied by slight wheezing, very perceptible to the observer, although the patient would consider himself quite free from dyspnœa.

There is another type of asthmatic, which may perhaps be distinguished as the gouty, in which the patient is full-fleshed, with excess of adipose tissue, especially about the abdomen, the general build suggesting the short-necked "apoplectic" subject, rather than the thin stork-like aspect of the victim of the more neurotic form of the disease. The distinction is one of considerable practical importance.

The Attack.—Patients with true spasmodic asthma may be seized at any time of the day or night, but more frequently the attack comes on at night after the first sleep. A certain feeling of oppression about the chest, attended perhaps with some

wheezing, may give warning of the approaching seizure; more rarely the attack comes on almost instantaneously, and without warning. The sense of oppression may partially awaken the patient or give rise to some disturbing dream, and he either starts up in a fright with the fit of dyspnœa full upon him, or more gradually awakens to the increasing difficulty of respiration.

A severe attack of asthma is, to the inexperienced, truly alarming to witness; the expression of face, pale, staring, anxious, and distressed; the mouth slightly opened, its corners twitched downwards with each brief effort at inspiration, whilst the neck muscles start forward in violent convulsive action. The shoulders are rounded, the body inclined forwards, and the hands rigidly grasp some firm object to fix the scapular and humeral attachments of the chest muscles. With the powerful inspiratory jerk thus effected the thorax is lifted *en masse*, but the deepening of the supraclavicular hollows and depressed lower thorax, sternum, and epigastrium, bear witness to the small penetration of air into the lungs in response to all this effort. Expiration is still more difficult, for the expiratory force is, so to speak, beaten in detail. The air-current in inspiration starts towards points of obstruction with a force proportional to expansion. In expiration, however, each infundibular current is obstructed whilst yet a feeble stream; the expiration is thus prolonged, laboured, and but feebly and gradually effected, when, without a moment's pause, the quick, short, powerful inspiratory jerk again takes place. The actual number of respirations per minute may not be increased, and is sometimes diminished; the pulse is, however, invariably quick, small, and often irregular and vacillating, and there may not infrequently be noticed a distinct hesitation or failure of the pulse wave with each inspiratory effort.

In earlier attacks, more especially, there is great restlessness and frequent change of posture, with a disposition to lose self-control in the desperation of air-hunger. But the experienced asthmatic assumes and maintains some favourite attitude. The severity of attack may last from a few minutes to many hours, but it will be observed that even in the most urgent cases there are intervals of partial relaxation, during which the breathing becomes more easy, to be followed shortly by full intensity of spasm.

Towards the close of the attack cough comes on, and with the expectoration of some viscid mucous pellets the dyspnœa is greatly mitigated. The body temperature during the seizure is depressed, cold sweats break out over the forehead, and the features become dusky and partially cyanosed. In very severe cases capillary hæmorrhages into the conjunctiva have been noticed (Walshe). The mind rarely becomes even for a moment clouded, and only in early cases does the patient sometimes lose that self-control which is of so much service to him in the struggle. The urine is generally abundant, pale, and of low specific gravity, and the solid constituents are lessened (Ringer). The subsidence of the attack is generally gradual, but sometimes almost sudden, with more or less expectoration, and the exhausted sufferer falls into a troubled sleep.

Physical Signs.—During the attack the thorax is semi-expanded, with but little movement in response to the respiratory efforts; the percussion note is resonant, and more or less of the emphysema type. Careful percussion is, however, impossible and useless. Auscultation reveals but little or no breath-sound, beyond a short wheeze in response to the sharp inspiratory jerk, whilst the expiratory murmur is wholly obscured by prolonged cooing sibili of varying note. The heart's impulse can be best felt, and sometimes strongly so, at or below the ensiform cartilage.

The amount of expectoration varies with the intensity and duration of the attack. The most characteristic expectoration consists of little pellets about the size of a pea, and "of the consistence of jelly or thick arrowroot, of a pale grey colour, of an opalescent transparency, and a saltish taste" (Salter). When carefully examined and unfolded these pellets will be found to contain casts of the finest bronchial tubes, constituting the peculiar formations known as Curschmann's spirals, which have already been described (p. 74). In almost all cases, too, on careful search, especially if the sputum has been kept for some hours before examination, Charcot-Leyden crystals will be met with (p. 75), sometimes within the spirals, sometimes free. As already indicated, neither crystals nor spirals can be regarded as pathognomonic of the disease. The asthmatic sputum contains in addition large numbers of eosinophile cells, and a considerable increase of these cells will also be found in the blood during the asthmatic attack:

their number may reach as high as 25 per cent. of the total leucocyte count.

Slight hæmoptysis is occasionally, but rarely, observed in severe cases of asthma. Where pulmonary œdema or congestion follows upon a severe paroxysm the expectoration is more abundant, consisting of a frothy, sometimes slightly blood-stained, mucus.

After a serious attack of asthma much prostration and fatigue are experienced from the severity and duration of the struggle and from want of food and sleep. These symptoms will soon be recovered from, but renewed attacks commonly ensue at short intervals, until at the end of a certain series the malady appears to be for a time exhausted.

More or less wheezing rhonchi generally persist for some hours or days, and in severe cases it is common to find at the bases some fine bubbling râles. The percussion signs and chest conformation at this period are those of acute pulmonary emphysema, varying in degree according to the severity of the attack. After a succession of attacks there is an amount of emphysema of the lungs, with œdema of their bases, and fatigue of the right ventricle of the heart, which requires some time and treatment to remove. From single attacks, even although very severe, the patient after a night's rest may feel quite restored. In other instances the attacks are brief, but return each night; these are usually cases in which the paroxysm is controlled by some remedy, and although jaded by disturbance of rest, such patients are able to pursue their daily work or pleasure.

Periodicity of seizure is usually a marked feature of asthma. The seizures may be daily, weekly, monthly, yearly, or at other tolerably regular intervals. A patient may suffer a series of daily seizures, and then enjoy a certain interval of freedom. It will be observed, however, with asthma, as with epilepsy, that as time advances the attacks, unless influenced otherwise by treatment, tend to become more frequent, although less severe, a number of minor seizures being interpolated between the more regular attacks, the gradually increasing emphysema rendering the intervals less and less defined.

In the earlier years of true spasmodic asthma patients are able in the intervals of attack to take part in sports and exercises, which make large demands upon the respiratory

powers. As the disease continues from year to year, however, it gradually entails other symptoms significant of definite pulmonary lesions; shortness of breath, and more or less wheezing continue through the intervals; the physical signs of emphysema remain permanent, and visceral congestion and associated dyspeptic symptoms become manifested. The heart especially suffers, its right ventricle becomes dilated, systemic venous fulness ensues, and, finally, œdema of extremities, abdominal dropsy, with enlarged and hardened liver and albuminous urine, supervene (see chapter on Emphysema, p. 271). In a certain number of cases of asthma, on the other hand, the lungs give way, an atrophic form of emphysema appearing, which proceeds to coalescence of vesicles with adjacent pulmonary fibrosis. Should this occur at one or both apices, the condition may readily be mistaken for chronic phthisis. In yet other cases pulmonary tuberculosis supervenes, with the onset of which the paroxysms of asthma may wholly cease.

The **Prognosis** of asthma proper is estimated by ascertaining the amount of physical damage which the patient has as yet sustained to lung and heart, for it is through complications referable to these organs that life is commonly shortened. Asthma *per se* never kills, for when the paroxysm actually threatens life the spasm yields; nor is the asthmatic prone to some of the diseases, such as cancer, tubercle, Bright's disease, which in others shorten life. His own peculiar malady is enough for him to contend with, and it enforces upon him a comparatively sheltered life. Asthma is, in fact, compatible with a life of medium length and of much usefulness, but of much suffering and self-denial. Family longevity should be inquired into in regard to prognosis. Infantile asthma, especially, if not definitely inherited, sometimes ceases at about puberty, and asthma of a gouty or influenzal origin is frequently cured by appropriate treatment.

Treatment.—There is no disease which is so extensively "quacked" as asthma. Persons who are the victims of the pure neurosis are amongst the most restless of mankind; their temperaments are often of the highly-strung, nervous type, and whilst they are gifted with much courage, endurance, and determination, they possess little faith, many friends, and much credulity. The practitioner who would guide these suf-

ferers must himself have clear opinions respecting the salient points in treatment, and must be patient in hearing and endeavouring to understand the experience of each individual.

The treatment of asthma may be considered under the following headings:

1. *Specific Treatment.*—When consulted by an asthmatic patient, we must recall what we have said as to the ætiology of the disease, remembering that in a proportion of cases it is the result of sensitisation to some particular protein. Careful inquiry must accordingly be made as to the patient's association with horses, cats, or cattle, and the use of feather beds or pillows. The question of pollen irritation must also be considered. In those cases especially in which the attacks have commenced in infancy, the possibility of sensitisation to some special food protein must be borne in mind, and sometimes the withdrawal of white-of-egg from the diet, or the substitution of goats' for cows' milk, will effect a great improvement. If the asthma is connected with bronchitis, the possible cause must be looked for in bacterial protein. After due observation such suspected factors may be scrutinised by the pathologist with the aid of the cutaneous test, and the particular protein, if any, discovered. If the malady be traced to a food protein, the special food must be eliminated from the diet, to be afterwards introduced tentatively and in regulated quantities, with a view to establishing immunity; if to an animal, pollen, or bacterial protein, desensitisation may be effected by gradually increasing doses of the appropriate vaccine.

2. *Climatic Treatment.*—This consists, in the first place, in the removal of the patient from those external surroundings which appear to have led up to his attack, to more favourable conditions of residence—*e.g.*, from a dusty to a pure atmosphere; from a cold, damp house or locality to a dry soil and a well-ventilated and cellared house, with no trees in the immediate vicinity.

To tell with precision what locality will suit a given individual with asthma is very difficult. There are idiosyncrasies in each case, and no asthmatic should burden himself with a house until he has first tested the locality by residence there for some time. There are, however, certain climates which are most likely to prove beneficial for asthma patients.

(*a*) Bournemouth, the St. George's Hill neighbourhood of Weybridge, and the Farnborough and Bagshot districts, with their sandy soil and pine vegetation, may be named amongst English localities as places which are under all circumstances preferable to cold, damp neighbourhoods, and which are peculiarly adapted for the purest forms of asthma in which the neurosis is most marked. Abroad, Arcachon may be similarly recommended during the winter and spring months.

(*b*) Torquay, St. Mary Church, Pau, Cimiez, Hyères, Mentone, Algiers, Algeciras, Grand Canary, and Santa Cruz (Teneriffe) may be mentioned as varied resorts adapted for winter residence for the mixed catarrhal forms of asthma.

(*c*) Experience teaches a certain number of asthmatics that a sea voyage does most for them, and in such cases the cure may best be thus started. This plan is especially suited for cases of dust and hay asthma, as also for those in which the first seizure has been traceable to a nervous system broken down by anxiety, overwork, or excesses.

(*d*) In young subjects, with as yet no marked emphysema, the pure rarefied airs of St. Moritz, Davos, Montana, and Arosa yield good results in winter, and in the hot months they may be resorted to with advantage by some older patients. In this connection we may perhaps add that we have known an asthmatic subject to remain quite well when residing for some years at Arequipa in Peru, at an altitude of 12,000 ft., but the attacks returned as soon as he again came to live at an ordinary level. Ilkley, Ben Rhydding, Dartmoor, Malvern, and Hindhead, are suitable situations in the summer season for convalescent asthmatics.

(*e*) A considerable number of asthma patients do best in towns, being chiefly those who have removed from more or less damp localities surrounded by trees. As a rule asthmatics should repair to large towns or seaside resorts in the late autumn, and perhaps the late spring.

3. *Medicated Airs and Baths.*—In cases of catarrhal asthma, in those of influenzal origin, and in those in which a gouty element or an association with eczematous eruptions or urticaria can be observed, a summer course of three or four weeks' duration, at Mont Dore, Aix-les-Bains, Allevard-les-Bains, or Dax, but especially at the first-named, will often be attended with long-continued benefit. Minute traces of

arsenic mingled with the vapours at the Mont Dore baths are regarded as answerable for their good effects, and at Allevard the sprayed air of the inhalation chambers is decidedly charged with sulphuretted hydrogen. This latter treatment is most adapted for the chronic bronchitic forms of the disease; at the other places probably the sweating of the skin and bronchial membranes constitutes the chief remedial factor. The course at one of these bathing resorts should be followed up by an after-cure at some more bracing place, such as Eaux Bonnes, Cauteret, Spa, or the Swiss mountains, or, in this country, at Ilkley, Ben Rhydding, Braemar, or other moorland districts of Yorkshire or Scotland. A course of Turkish baths for those who cannot go abroad sometimes proves of great service. We have in some instances seen much benefit from the use of the compressed-air baths at the Brompton Hospital (see p. 281), but we can only mention this as an empirical fact.

4. *Regulation of the Digestive Function.*—Not less important than the selection of a suitable climate is the careful regulation of the digestive functions of the asthmatic. In not a few cases the cause of the attacks is associated with the habitual ingestion of an excessive quantity of food, and if this be diminished the frequency of the paroxysms will be greatly curtailed. In other instances the exciting cause is some error in digestion which requires careful regulation and attention, and in all cases the digestive function ultimately suffers, and reacts unfavourably upon the spasmodic troubles. Very slow eating should be strictly enjoined, and the staple food, including a moderate quantity of meat, should be taken at the midday meal, only the lightest possible diet being allowed later in the day. In some cases where farinaceous foods ferment, a more nitrogenous dietary, approximating to the "Salisbury" system, may be recommended. In all cases it is wise to substitute for bread with the meat meals unsweetened rusks, or Huntley and Palmer's breakfast biscuits, or baked toast. In cases in which there is excess of uric acid or urates, and a disposition to flatulent dyspepsia, cutaneous eruptions, and so forth, sweet wines, raw fruits, sugar, cooked butter, and pastry should be excluded from the dietary. An alkaline bitter may often be taken twice a day with great advantage when the tongue is red and coated, bismuth being added when there is gastrodynia.

The liver and bowels require careful attention, very small doses of mercurial being useful from time to time, whilst in these, as in all neurotic subjects, large doses of this drug are positively harmful. Calomel, given occasionally in half-grain doses at night, is especially valuable. A sulphate of soda saline should be taken in hot fluid in the morning after each dose of mercurial. In other cases a teaspoonful of Pulvis rhei co. at bedtime will prove beneficial. If the rectum be loaded, relief should be effected by enemata rather than by violent aperients.

When the tongue is fairly clean, or at all events when the practitioner is satisfied that the *primæ viæ* are acting efficiently, a course of arsenic is often of great service.

5. *Attention to Other Sources of Peripheral Irritation.*—What we have urged in regard to the digestive functions applies also to other organs, and if any definite source of peripheral irritation exists, it should receive attention. The condition of the nasal cavities should always be carefully investigated, since improvement, sometimes even permanent cure, has resulted from the removal of a polypus or the treatment of sinus suppuration or some other obvious defect. In the absence of any such gross lesions we should hesitate to advise prolonged or serious nasal treatment, for although transitory amendment may follow upon the shock or distraction of any operative measure, no benefit of a permanent character results unless there be some definite condition to be amended. The application, however, of the galvanic cautery to various points upon the nasal mucous membrane, especially of the septum, not uncommonly gives temporary relief, and as the procedure is a simple one, it may be tried in obstinate cases.

Treatment of the Paroxysm.—Most commonly the practitioner makes his first acquaintance with the asthmatic patient when the paroxysm is at its height. A few questions as to duration of attack, preceding attacks, and circumstances and symptoms immediately antecedent to the present seizure, will suffice to direct a rational and safe treatment.

When irritating matter is present in the stomach, whether in the shape of undigested food or irritant catarrhal mucus, an emetic of twenty grains of ipecacuanha, or a subcutaneous injection of $\frac{1}{10}$ to $\frac{1}{6}$ grain of apomorphia, will give prompt

relief by its removal. Warm water with a little carbonate of soda should be given to encourage a thorough clearance of the stomach.

In cases of catarrhal asthma, during the paroxysm, ten minims of ipecacuanha wine, with fifteen minims of ethereal tincture of lobelia, given every half-hour for two or three doses, then every hour or two hours, will often prove serviceable. The liquid extract of grindelia robusta (U.S.P.) in fifteen-minim doses may be sometimes substituted for the lobelia.

In the choice of sedative remedies, a careful judgment must first be arrived at as to the amount of tubal catarrh and secretion present. The well-known "cures" of asthma appear to owe their efficacy partly to an evacuant, partly to a sedative, action. The fumes from burning the "Himrod," "Potters," "Green Mountain," "Chester" and other similar powders, first excite more or less cough, after which, and especially if expectoration be effected, the spasm yields. A powder containing four drachms of powdered stramonium leaves, two drachms of powdered nitre, a similar quantity of aniseed, and five grains of tobacco is a very efficacious combination, much used at the Brompton Hospital. A teaspoonful of this powder should be made into a conical heap on a plate, lighted at the summit, and the fumes inhaled through a large inverted funnel. In the more purely nervous forms of asthma, when as yet there is no secretion present, sedative remedies must be used.

By closely shutting the room and filling it with the fumes of nitre* the asthmatic sufferer will sometimes gain relief and rest. Salter considered nitre thus used a powerful sedative, but it must be confessed that its mode of action remains obscure. Its efficacy, however, in certain cases is unquestionable.

In other cases speedy relief has been obtained by spraying the nasal cavities with a solution of adrenalin chloride, 1 in 2,000, or by the subcutaneous injection of 1 to 5 minims of the 1 in 1,000 solution, the adrenalin paralysing for a time the nerve endings, thus allowing the bronchioles to dilate. Extract of the posterior lobe of the pituitary body, which is

* Take four ounces of nitrate of potash dissolved in half a pint of boiling water, pour out into a soup plate, immerse in the solution thick blotting-paper, dry, and cut in squares of four inches. A little chlorate of potash, about half a drachm to the ounce, may sometimes be usefully combined with the nitre.

stated to act like adrenalin, but to have a more lasting effect, is helpful in some patients.

In the full height and intensity of attack nitrite of amyl, iodide of ethyl,* or chloroform, may be inhaled with temporary advantage, and the true spasmodic nature of the affection will be well demonstrated by the complete subsidence of the sibilus under the influence of chloroform. The effect of these drugs is, however, as a rule very evanescent, and their use is only desirable to mitigate extreme symptoms. Strong coffee sometimes distinctly relieves spasm, and "eupnine" a solution containing caffeine and iodine, may be given in drachm doses in water, and repeated in half an hour, with a like result. Nitroglycerine and nitrite of sodium have also been recommended.

Joy's cigarettes and Savory and Moore's datura tatula cigarettes will, provided the patient can inhale the smoke into the lungs, in many cases give great relief to the urgent symptoms, their efficacy being due to stramonium and, probably, some opium combined. Another remedy much used by the public is "Dr. Tucker's Asthma Specific," a liquid which is inhaled as a very fine spray from a vaporiser or atomiser, and which is often effective in relieving the spasm. An analysis of its contents, made by Dr. Willcox,[10] showed each fluid ounce to contain cocaine 2·28 grains, atropine 0·87 grain, sodium nitrite 15·25 grains, a certain quantity of balsam or gum-benzoin, between 20 and 30 per cent. by *volume* of glycerin, together with traces of mineral ingredients, vegetable colouring matter, and so forth. This mixture when inhaled often gives great relief, but it contains potent drugs, and its use has sometimes been attended with disquieting symptoms. It should be employed, therefore, with caution, the tendency to its use becoming a habit being also remembered. An analogous remedy is the Nebula hyoscinæ co. of the British Pharmaceutical Codex.

In severe cases an almost unfailing remedy is morphia used subcutaneously in doses of from $\frac{1}{6}$ to $\frac{1}{2}$ grain. Morphia is a remedy which, from the immediate relief it affords, is apt to be given somewhat recklessly, and to be called for peremptorily by the patient, without counting the cost in after consequences. The drug should be used with caution, for

* Convenient five-drop capsules of this drug, enclosed in silk, similar to those of nitrite of amyl, can be obtained.

not a few casualties have arisen from its employment. In catarrhal cases it should not be employed, and in the most purely neurotic cases, for the relief of which it is best adapted, it is a demoralising remedy. In fact, the employment of morphia in asthma is exactly comparable to its use in neuralgia; prompt in relieving, it lowers nerve tone, hampers secretion, and increases liability to recurrence. The tendency of the patient is to have recourse increasingly to its use for trivial attacks, until custom renders a large and dangerous dose necessary. In certain cases the drug is required; they are exceptional, however, and the most strenuous efforts should be made by the practitioner in such cases to remove his patient out of the conditions to which the asthma appears attributable. Hypodermic injections of atropine, $\frac{1}{100}$ to $\frac{1}{80}$ grain, either alone or in combination with morphia, are sometimes useful. More recently heroin has been used with good results, in doses of $\frac{1}{12}$ to $\frac{1}{6}$ grain of the hydrochloride, given subcutaneously and repeated in an hour if necessary. But here again there is a danger of the use of the drug degenerating into a habit.

Chloral in full doses of ten or twenty grains every four or six hours is also efficacious in pure asthma, and it may be used in the catarrhal forms; its administration, however, requires strict medical supervision and very precise directions. It may sometimes usefully be given in small doses in combination with iodide of potassium and other drugs.

In cases of catarrhal asthma especially, and in some other less-defined cases, one of the most valuable remedies is iodide of potassium, in combination with stramonium. The iodide is especially indicated in those cases in which there is a nightly paroxysm, but in which there is also a perceptible dyspnœa and wheezing throughout the day. Three to five grains of iodide of potassium, with $\frac{1}{4}$ grain of extract of stramonium, should be given every three or four hours during the day, *e.g.*, 8 a.m., 12 noon, 3 p.m., 6 p.m., 9 p.m., so as to administer some fifteen to thirty grains of the salt and one to two grains of the extract by bedtime. The addition of small doses, $\frac{1}{24}$ grain, of apomorphia to the mixture is sometimes helpful. After a day or two when the patient suffers slight iodism, and the physiological effect of the stramonium upon pupil and throat becomes apparent, the medicine may be continued in half doses. In the many cases in which this line of treatment proves valuable

it should be commenced on the first approach of asthma phenomena. Another remedy which we have found of use in warding off asthmatic attacks is "Grindeline," which contains small doses of grindelia robusta, euphorbia, liq. trinitrini, and potassium iodide. A drachm in water should be given every six hours. In some cases in which asthma has been contracted in malarious climates, or in which a neuralgic element is traceable, quinine may be usefully given in combination with iodide of potassium. Five grains of salicylate of quinine in pill (with citric acid) may be taken three times a day, either at separate times or with each dose of iodide.

In hay asthma the use of pollantin, the specific serum produced under the direction of Professor Dunbar[9(1)] (see p. 248), has been found of some value. The dry powdered serum should be sniffed up into the nose on first waking in the morning, and again several times during the day, whenever the slightest sign of an attack appears. The pollantin, whether in the dry or liquid form, should also be applied to the eye if necessary. General precautions, such as sleeping with the windows closed during the hay-fever season, should not be omitted. In certain cases the results to the hay fever and the hay asthma from this specific treatment have been gratifying, the patient having been, for a time at least, freed from his complaint. In other instances failure has resulted, or some amelioration only obtained.

Should the patient come under observation during the winter or spring, a course of preventive subcutaneous inoculation with "Pollacine" (a liquid extract containing the pollen protein) may be tried,* it having been first shown by a positive ophthalmic reaction that the patient is a sufferer from true hay fever. In certain cases considerable relief is thus given during the succeeding summer.

Relief from the itching of the eyes and the sneezing which are so often associated with hay asthma may be obtained by inhaling from a handkerchief a solution containing camphor ʒss., menthol ʒi., paroleine ℥i., or by lightly plugging the anterior nares with cotton-wool moistened with a solution of four grains of hydrochloride of cocaine in one ounce of a 1 in 10,000 solution of adrenalin hydrochloride. The plug

* Outfits, prepared in the Inoculation Department of St. Mary's Hospital, are supplied by Messrs. Parke, Davis and Co.

should be inserted in the early morning on rising, and again during the day, whenever the earliest signs of an attack are noticed. If any highly sensitive spots be discoverable in the nasal mucous membrane, they may be cauterised. Attention must be paid to the digestion and the general health, and in some cases a sea voyage may be prescribed with advantage, or a short residence at one of our seacoast places, such as Yarmouth or other exposed marine station.

Alcoholic stimulant should only be given in asthma when necessary as a restorative and in small doses. Strong coffee is of great value as a restorative after a severe paroxysm, and, as we have seen, in some cases it distinctly relieves spasm. Caffeine (two or three pills of 2½ grains each, made with glycerine of tragacanth) may be taken for the same purpose. After an attack of asthma, especially if it has been severe and has left any pulmonary œdema behind, it is a good practice to give small doses of digitalis (five to ten minims three times a day) to restore tone to the heart and small vessels. At this period arsenic is a most valuable nerve tonic.

The digestive system now requires careful attention, since the secretions will necessarily have become disordered by the drugs used for the relief of the paroxysms, and by the venous congestion of viscera attendant upon the impeded pulmonary circulation. It is probable, too, that the function of the pneumogastric nerve becomes seriously enfeebled for a time by the attack and the remedies employed. Some pepsin and hydrochloric acid after food, in combination with nux vomica or quinine, given an hour or two before food, will prove serviceable. The most careful consideration must immediately be given, however, as to whether the patient can be moved to a more suitable locality, and this may in some cases be wisely effected, even in the midst of an attack.

The Use of Vaccines as a Prophylactic Measure.—In some cases of asthma of a distinctly catarrhal type, which begin with "colds," influenza attacks, and the like, and cases in which there is chronic bronchial catarrh with asthmatic paroxysms, the use of an appropriate vaccine is attended with decided benefit. The vaccine should be carefully prepared from the sputum of the subject, the M. catarrhalis being in many cases the principal organism, associated perhaps with the staphylococcus, streptococcus, pneumococcus, or Pfeiffer's bacillus.

The vaccine is given at first in moderate doses twice a week, and gradually increased according to the degree of reaction, the larger doses being prescribed at first once a week or ten days and then once a month. The treatment requires to be continued for several months, and perhaps renewed from time to time, selecting a few weeks precedent to the times at which the patient is most liable to his attacks. It is important to remember that in the use of these, as indeed of all vaccines, a period of quietude of several hours should succeed each injection. It is thus convenient to give the injection about teatime, and to enjoin that the patient remains in bed until noon of the following day, by which time the degree of reaction, if any, will have been noted.

We have already enjoined great caution in the use of sera therapeutically in asthmatic subjects, whom it is well first to desensitise (see p. 319).

We may, perhaps, conclude this chapter with an account of the two following cases. The first is an example of bronchitis and asthma produced by the inhalation of rosewood dust, in which the effect of treatment was very striking.

William C——, aged thirty-eight, residing in London, was a man of temperate habits, married, with one child living; his wife had had one miscarriage, but there was no history of syphilis. He had been for twenty years employed as a fret-cutter. Four years previous to admission into the Brompton Hospital he had had an attack of bronchitis, and since that time he had suffered constantly from cough, with suffocative attacks nearly every night. Patient had never had hæmoptysis, but had lost flesh considerably. His father died of rheumatic gout, and his mother of dropsy. There was no phthisis in the family. On admission patient complained of cough, which became worse, and was attended with severe attacks of dyspnœa at night. Expectoration moderate; occasional night-sweats; appetite poor; bowels constipated.

He was a tall and fairly well-built man, with a somewhat suffused countenance and breathless look. The chest was well formed, but expansion with inspiration was impaired. The percussion note over the front of the chest was hyper-resonant, the resonance extending on the right side a hand's-breadth below the nipple, on the left side over the normal area of heart's dulness, and inferiorly to the costal margin. Diffused sibilant râles were audible front and back, and at the posterior bases some mucous râles were heard. When seen on January 26 the patient had been in hospital three days, and was

suffering, as before admission, from nightly attacks of dyspnœa and from troublesome cough. He was on the ordinary full diet of the hospital. The "Mistura Potassii Iodidi cum Stramonio" of the hospital Pharmacopœia, containing three grains of iodide of potassium, ¼ grain of extract of stramonium to each dose, was now ordered to be taken at 12 noon, 4 p.m., 8 p.m., and 12 midnight, the diet remaining the same, and from the next night he had no serious dyspnœa. On January 31 a note is entered: "Patient feels much better, and dyspnœa almost gone. Cough easier." February 3: "Has not felt suffocating sensation for the last six nights." He continued the mixture, however, for a month, and then took it in half doses for another fortnight. On February 8 respirations were free and unaccompanied by râle, and by the end of the month the cough had disappeared. On leaving the hospital at the beginning of March his weight was 9 stone 2 pounds, an increase of seven pounds since his admission.

It should be observed in this case, as in many others, that mere rest from his dusty occupation did not suffice for the patient's recovery, although doubtless, had he remained in pure air a sufficient length of time, he might have recovered without treatment. His attacks had the paroxysmal character peculiar to asthma, coming on towards the small hours of the morning—*i.e.*, after a certain period of repose—whilst the breathlessness and cough of the bronchitis and attendant emphysema were constant through the day. Having regard to the period at which the dyspnœa became distressing, the stramonium and iodide of potassium mixture was so ordered that by midnight he had taken in the course of twelve hours a grain of stramonium extract, and twelve grains of the iodide. The effect, as we have seen, was striking and immediate.

The mechanism of the dyspnœa was obvious. The man had an unduly secreting, and probably a somewhat thickened, bronchial tract, with great irritability of the bronchial muscular apparatus, and constant tendency to spasm of the tubes. At a certain time after repose secretion would accumulate and give rise to spasm. The hypersensitiveness upon which the bronchial spasm depended was at once lessened by the stramonium, whilst the iodide had a more permanently alterative action upon the mucous membrane and its secretion.

The patient left the hospital well, but after a time, failing to find other work, was reduced to the necessity of recommencing his old employment, and soon had a return of all the old symptoms, for which he was treated elsewhere by ordinary remedies (ether and expectorants) without avail. We have, however, since seen him in fairly good health, having for some time abandoned his former work.

In the trade of fret-cutting the operator has constantly with his mouth to blow away the fine wood-dust that collects upon his work, and thus necessarily inhales much of the dust. The patient above referred to stated that he could distinguish by the taste the different kinds of wood, and he found rosewood (the taste of which he com-

pared to cayenne pepper) to be the most irritating. Walnut-wood was more astringent and bitter, but less irritating.

Asthmatic paroxysms are not infrequently associated with bronchial catarrh, and the following is the epitome of a case in which a chronic bronchial catarrh of gouty incidence, with heightened blood-pressure and some degree of arterio-sclerosis, was so associated, the special feature of the case being the constant presence in the sputum of numerous casts varying in size from the smallest filaments to those having a diameter of $\frac{1}{8}$ inch (Plate XIV.).

The case was that of an unmarried lady, aged sixty-six, seen by Sir Douglas Powell, in consultation with Dr. George Smith of Henley, in July, 1919. There was no inheritance of an asthmatic kind, and, although for many years subject to repeated colds, she had never had an attack until her present illness. This began in January, 1914, with attacks of violent sneezing, which recurred every few days without apparent reason and were not influenced by remedies. Between the attacks her health was good. The spring and early summer months were spent on the Italian Riviera, at Naples, and in Rome and Florence.

Her first attack of asthma occurred at Naples late in the spring, and continued in Rome and Florence. The attacks were attended with complete obstruction of the nasal passages, presumably from congestion of the mucous membrane covering the turbinate bones, and with difficulty in speaking or swallowing during the attacks. Severe paroxysms of coughing followed. The worst attacks occurred between 4 and 8 a.m.; those less severe between 4 and 8 p.m. At Florence she was laid up for six weeks with bronchitis, and on convalescing she was passed on to Ems, where she had a relapse. Finally she suffered a terrible ten days' journey home at the commencement of the war. Vaccines were repeatedly employed on the Continent, prepared from the sputum. There seems to have been some uncertainty as to the nature of the microbe, and no relief was derived from the vaccines. From 1914 the ashmatic paroxysms became increasingly frequent, and the only remedy that gave relief was adrenalin, 5 minims subcutaneously of 1 in 1,000 solution. She had been accustomed to take this four or five times daily for relief of the paroxysms. In May, 1919, she underwent an operation, quite unconnected with the air passages, and, as is so common in asthma, her dyspnœic troubles entirely cleared away for seven weeks!

During the three weeks previous to the consultation in July the symptoms had been gradually returning, and in the past fortnight there had been several hours of violent sneezing with nasal obstruction and with threatenings of the bronchial asthma—symptoms, in fact, identical with those with which her illness commenced in 1914.

PLATE XIV

CASTS FROM THE BRONCHIOLES FROM A CASE OF BRONCHITIC ASTHMA

The drawing shows in their actual size numerous small bronchial casts of the thickness of thin string or cotton, white and forked, forming collections like bunches of thin white ribbon. They were expectorated by a lady, aged 66, of gouty diathesis, who suffered from asthma associated with bronchial catarrh and raised blood-pressure. The case is described in the text (p. 266).

PLATE XIV

Casts from the Bronchioles from a Case of Bronchitic Asthma.

To face p. 266.

At the time of the consultation the expectoration was considerable in quantity, mucoid and frothy, and containing numerous tangled strings or casts, which in the earlier part of her illness were sometimes "as thick as pencils," but were now mostly small, quite white, forked, and of the thickness of thin string or cotton, forming collections rather like bunches of little white ribbons. Fearing she might have a spasm during examination, the lady had taken 5 minims of adrenalin twenty minutes previously. The breathing was quiet, and there was no cough during the interview; the pulse 84, of notably high tension, the blood-pressure being found to be 95 diastolic and 205 systolic. The heart's apex beat was slightly to the left of the normal, the sounds clear, and the second sound heavy and accentuated. There was some slight sclerosis of the vessels. The lungs were a little emphysematous, and over the bases posteriorly fine crepitating râles were heard, which diminished, but did not disappear, after several deep inspirations.

The finger-ends were enlarged with gouty arthritis, and it was elicited that the lady had always been gouty and had had several distinct attacks of gout in the feet.

Dr. Smith subsequently made several examinations of the blood-pressure apart from adrenalin influence, and found the average pressure about 165; sometimes over 180; once as low as 140.

The patient had lately been living in rooms, with stuffy furniture and insufficient air, and a removal to Peppard Common, with wheel-chair and quiet exercise, together with iodine and eliminative treatment, proved beneficial. On her return home, however, although continuing the better hygienic conditions, the symptoms relapsed, but with less severity. The drawing in Plate XIV. was taken from a specimen of sputum expectorated in January, 1920. The report stated that "the sputum consists of mucus in which are whitish strings of varying size. These consist of mucoid material, with a tough, tenacious, possibly fibrinous, core, in which are a few bronchial cells and abundant leucocytes, but no alveolar cells. The prevailing organism seen in a stained film was a Gram-negative bacillus of coliform appearance, but there were also a very few cocci."

The case was complicated with epithelioma quite unconnected with the pulmonary organs. During the last few weeks of her life, apparently under the influence of salicylate of soda, the patient was again relieved of all asthmatic symptoms. It must, however, be recorded that during this time she had several smart hæmorrhages from the malignant growth, in one of which she died in the spring of 1920. There was, as shown on two examinations, no marked association with bacterial organisms, and consequently, as one might expect, vaccines proved of no avail.

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CHAPTER XVI

PULMONARY VESICULAR EMPHYSEMA

PULMONARY vesicular emphysema may be defined as a dilatation of the air-cells and infundibula of the lungs, with antecedent or associated atrophic changes, or, more briefly still, as a dilatation of the lung proper with textural atrophy.

In its acute form this disease may affect persons of any age, but is especially apt to occur in children as a complication of whooping-cough or capillary bronchitis. In its chronic and more permanent form it affects most commonly persons in middle or advanced middle life. Atrophic or senile emphysema, which scarcely merits consideration as a variety of the disease, affects only aged people, and is, indeed, but a part of the senile state.

PATHOLOGY.—The perfectness of the function of respiration consists quite as much in the power of contracting as in that of filling the chest, and it is this power of contracting the chest that is lost in emphysema. The lungs, having lost their reserve elasticity, no longer tend further to contract at the completion of expiration; nay, expiration is never completed; the thoracic parieties and diaphragm, instead of being drawn inwards by the traction of the lungs, recoil simply to their position of repose, and oppose their dead weight to the inspiratory muscles instead of aiding the action of these muscles by their elastic rebound. Hence, in extreme emphysema the inspiratory act, commencing at the point where in health calm inspiration would end, has to overcome (1) what remains of the elastic resistance of the lungs; (2) the inertia and the resistance of the parietes, instead of, as in health, having to deal with the elastic resistance of the lungs alone, and being in this work aided by the outward spring of the ribs (see p. 9). Consequently, in marked cases the breathing is always forced, and more or less difficult.

Let us now briefly recount the conditions present in emphysema, making such additional comments as seem called for.

1. The lungs in all cases of decided emphysema are permanently expanded to about the position of ordinary inspiration, their elasticity being, so to speak, relaxed to this point. The enlargement commences, as pointed out by Rindfleisch,[1a] in the infundibula, and, according to Dr. Arthur Keith,[2] affects this portion of the lung chiefly. As a result of these changes, the pulmonary vascular system becomes lengthened. In acute emphysema loss of tone from repeated over-distension, *e.g.*, during paroxysms of coughing, is the chief defect present, and, in young persons especially, is speedily recovered from.

2. The texture of the lung is to a greater or less extent impaired.

In cases of long-standing emphysema resulting from repeated attacks of asthma or bronchitis, what may be regarded as the second stage of the disease is entered upon; and the lung suffers in nutrition and elasticity. Many of the small bloodvessels become obliterated, withering backwards from their capillaries, a result due in part to primary degenerative changes in the capillaries, and in part to atrophic changes consequent upon the narrowing and stretching to which the vessels are subjected as the lung enlarges. Concurrently with these changes the alveoli atrophy, the portions between adjacent infundibula or alveolar spaces become thinned and finally give way, and the spaces here and there coalesce to form blebs or small cavitations, with thread-like remnants of vessels crossing them, resembling in miniature the trabeculæ of tuberculous cavities. Where the disease has resulted from oft-recurrent catarrhs, commonly with a history of a distinct attack of bronchitis at the commencement, ill-developed fibrous tissue, the result of repeated and long-continued congestion and nuclear proliferation in the submucous layer, toughens the lung texture, assists in impairing elasticity, and partakes in the subsequent degeneration. Similar changes occur in the local emphysema developed about old tuberculous centres in the lung.

In cases of constitutional or hereditary origin the atrophic changes, whether originating in fatty degeneration of the pulmonary epithelium, or of that of the vessels of the lungs,

or in a primary degeneration and rupture of the elastic fibres —and on these points authorities are by no means agreed[3]— would appear to precede or accompany the catarrhal phenomena. This impairment of the texture of the lung renders any dilatation, however induced, more or less permanent. In these cases of primarily impaired lung texture, enlargement of the chest to the limits of thoracic resilience takes place quite insidiously, and the chest thus assumes permanently, by imperceptible degrees, the position of moderate inspiration. It must not be forgotten, too, that the ribs themselves, and especially their cartilages, often prematurely undergo textural changes of a degenerative kind, the result of which is an increased rigidity and a straightening of the rib arch, and so an enlargement of the thorax.

3. In consequence of the relaxed elasticity of the enlarged lungs, they no longer exercise any traction upon the mediastinum except during inspiration. Hence an important, because constant, aid to the return of blood to the heart is lost.

Many authors go farther than this, and hold that the large lungs in emphysema are, so to speak, pent up in the chest, and exercise pressure upon the heart which lies between them, and upon the ribs and diaphragm which enclose them. A little reflection and clinical observation will, however, render it clear that this supposed pressure of the lungs upon the surrounding parts is, if not impossible, of infinitely rare occurrence and minute in degree. The enlarged thorax, flattened diaphragm, and lowered heart, are all phenomena due to defective recoil of the lungs, not to their active distension, and are to be observed in health on the lungs being inflated during deep inspiration, a position which is retained in emphysema.

4. In addition to the negative impediment to the circulation referred to in the preceding proposition, there is a positive impediment in the stretched, and in part obliterated, capillaries of the lungs.

5. In consequence of the two last-mentioned conditions, the venous system is overfull, and the overworked right heart thickens. The increased power of the right ventricle at length becomes, however, inadequate to contend against the ever-present resistance to the pulmonary circulation, a resistance

which is from time to time accentuated during attacks of bronchitis or asthma, and the whole venous system becomes engorged. With general venous congestion the return of blood from the coronary veins is impeded, and the heart muscle suffers in nutrition, undergoing fibroid degeneration. This in its turn leads to imperfect contraction of the ventricles, and causes them to yield gradually before the heightened blood-pressure. Hence follows a greater degree of venous stagnation and engorgement, especially in the hepatic and portal systems. Venous hæmorrhages tend to occur, and œdema, commencing in the legs, sooner or later sets in.

6. Another effect of the relaxed state of the lung in extreme emphysema is to interfere with that condition of permanent patency in which the small bronchi are normally held by the constant traction of the elastic lung upon them from all sides. In emphysema this traction becomes neutralised during expiration, and in more advanced cases collapse of the bronchioles must then occur, thus accounting for the laboured and obstructed character of that portion of the respiratory act.

7. Amongst the consequences of emphysema, those which are of the nature of *compensation* must not be forgotten. So ready are the adaptations of our economy to altered circumstances, that it may be said that emphysema alone does not kill. (*a*) As the capillaries become partially obliterated, fresh communications are opened up with the neighbouring vessels corresponding to them by the formation of anastomotic loops, or by the widening of communications already existing. The pulmonary and systemic veins, we may remember, communicate normally on the walls of the smaller bronchi, and with the formation of pleuritic adhesions, so common in this disease, loops of new vessels connect the pulmonary with the systemic circulation; and especially along the insertion of the diaphragm and margins of the sternum, fringes of enlarged vessels mark the connection between the two circulations. Thus, in some measure the pressure of the pulmonary circulation is eased. (*b*) The hypertrophy of the right ventricle of the heart above alluded to is, up to a certain point, of a strictly compensatory nature, serving to overcome by augmented power the increasing impediment to the pulmonary circulation. Thus, for a time at least, equilibrium is maintained, to be disbalanced again and again, however, by intercurrent

attacks of bronchitis or other cause of overstrain, until at last the limits of compensatory recuperation are passed.

ÆTIOLOGY.—When we come to inquire how this over-expansion of the air-vesicles is produced, we are met by various explanations, no one of which alone is sufficient to account for all cases. Sir William Jenner[4] demonstrated that *expiratory effort* during straining or coughing, particularly the latter, is an efficient cause of general emphysema, those portions of lung which are least supported, viz., the apices and anterior margins, and also the parts corresponding with the comparatively yielding intercostal spaces, becoming first affected. But with the production of emphysema in these portions of the lungs, a shifting of the relationship between the lung and the thoracic surface takes place, and parts which were originally in apposition with the ribs come to be opposed to interspaces, and in their turn yield before the distending force of air pent up and compressed during cough.*

Following this view, it has been believed that the playing upon wind-instruments is an occupation likely to lead to emphysema from the expiratory efforts involved. The experience at the Foundling Hospital would, however, suggest that in young and healthy subjects this is not the case. At this institution many of the boys are trained for the regimental bands, and Dr. J. C. Swift, for many years Medical Officer to the hospital, informs us that over a period of more than twenty years only one case of emphysema developed among nearly 600 boys so trained. The subsequent health also of these boys in the army has been good.

Other authorities, commencing with Laennec,[5] have held that *inspiratory pressure* is the chief factor, and in the production of local emphysema it is undoubtedly an important agent. Thus, if one lung be disabled or bound down by some inflammatory process (as old pleurisy or chronic pneumonia), the other lung perforce becomes more capacious; whether this extra capacity shall be derived from true hypertrophy or mere dilatation (emphysema) depends upon the nutritive vigour of the patient. During bronchitis certain of the air-

* Sir William Jenner (Reynolds's *System of Medicine,* vol. iii., p. 478) refers to Mendelssohn as having in a paper, "Der Mechanismus der Respiration und Circulation," in 1845, anticipated him in his view respecting emphysema. The authorship and advocacy of the view in this country rests, however, with Sir W. Jenner.

tubes may become occluded by mucus, and the inspiratory force then operates as a distending power upon the remaining portions of the lung until the deficiency in air-space is compensated. Thus, in the words of Rindfleisch,[1b] who adopted the late Sir William Gairdner's[6] inspiratory theory in regard to the production of emphysema, "during the antecedent bronchitis, first one, then another bronchial tube is plugged with secretion, and so first one, then another segment of the lung is subjected to an abnormal degree of [inspiratory] distension," until at length general emphysema is produced. Dr. Gee,[7] in his Lumleian Lectures, also advocated the inspiratory as being the chief factor in the production of emphysema.

An impartial view of the matter renders it clear to us that each of these theories is valid and applicable to a considerable number of cases, now one and now the other predominating. But both inspiratory and expiratory theories imply the presence of some pre-existing bronchitis, or some local lesion disabling a portion of the lung, conditions which are wanting in a considerable proportion of cases.

In truth, the emphysema frequently precedes the bronchial affection, although it is subsequently aggravated by the attacks of bronchitis; and admitting with the late Dr. Greenhow[8] and others that a *failure of nutrition* is, in a large proportion of cases, the predisposing cause of emphysema, we need go but little farther to explain the occurrence of that disease in its earlier grades. The effect of damaged nutrition of the lungs is to impair their elasticity and traction upon the thoracic walls. As we have elsewhere shown (see p. 11), the thoracic resilience tends to expand the chest from 1 to 3 millimetres in each direction; in proportion, therefore, as the lungs lose elasticity, they yield to this resilience of the thoracic wall, and to the weight of the abdominal organs dragging upon the diaphragm, and thus the lung is expanded to such a degree that any further extension will suffice to cause symptoms of dyspnœa. Calcareous degeneration affecting the cartilages and ribs, referred to by Freund,[9] causing loss of elasticity of these parts, may lead, for reasons already given, to a rigid enlargement of the chest cavity. This change takes place *pari passu* with, or may even precede, the loss of pulmonary elasticity, and thus plays a part in the ætiology of the disease.

It is from this latter view of its mechanism that one can appreciate the fact that in emphysema the chief defect consists in the lessened power of contracting the chest, and thereby emptying the lungs. The act of expiration is never completed; there is too much residual air constantly in the lungs, and inspiration is short and jerking, the act commencing where it should end.

The force of expiration, as estimated by the manometer, is remarkably lessened in emphysema, whilst that of inspiration remains normal, or may even be increased (Waldenburg),[10] the relationship between inspiration and expiration being thus, in this respect, the reverse of that obtaining in health. The vital capacity of persons with emphysema is, as might be supposed, greatly curtailed.

Varieties of Pulmonary Vesicular Emphysema.

The distribution of pulmonary vesicular emphysema may be local or general, and, as above shown, it may vary in degree between wide limits.

Local Pulmonary Vesicular Emphysema.—This may be met with as a dilatation of the lung, accompanied by more or less atrophic changes, around old cicatrising nodules of disease, contracting cavities, or points of pulmonary collapse. The site of the emphysema is here determined by that of the primary lesion, on which its presence mechanically depends. Whilst the effect of the emphysema is clinically to mask the physical signs proper to the consolidation, it brings no compensatory advantage to the patient, since the emphysematous portions of the lung are deficient in respiratory function.

Again, it sometimes happens that a whole lung is disabled by destructive disease, by permanent collapse from long-continued fluid pressure or interstitial fibrous growth. In these cases it is inevitable that the opposite and originally unaffected lung shall enlarge, and fortunately in many instances this enlargement is really of the nature of compensatory hypertrophy; there is, that is to say, greatly increased mobility of the side, the respiratory murmur is loud and puerile over the whole extended area of the lung, and the patient's breathing powers are fairly maintained. We may, therefore, safely assume in such cases increased function corresponding with increased size, which is at least the clinical

definition of true hypertrophy. On minute examination we believe that a corresponding development of lung texture, with increased blood-supply, and an absence of the atrophic changes of emphysema, would be found.[11]

In other cases, however, in which the lung disablement has occurred in a subject of broken constitution, whether from hereditary tendency, prolonged fever, faulty habits, or comparatively advanced age at the time of attack, the "sound" lung, whilst enlarging to the clinical outline and pattern of hypertrophy, does not yield to auscultation evidence of vigorous breathing, but a feeble *diluted* breath-sound, wanting in concentrated vesicular quality. The expanded side lacks mobility, and rustling crepitant sounds may be heard at different points, especially near its anterior margin. The patient has not the signs of improved aëration, he presents a livid tinge about the peripheral parts and extremities, and his enlarged lung has brought no corresponding relief to his breathless condition. Here we have a dilatation of the lung mechanically induced, and attended with atrophic changes, a condition which constitutes emphysema.

General Pulmonary Vesicular Emphysema — 1. *Large-Lunged Emphysema.*—This is the condition of typical symmetrical emphysema, to the description of which this chapter is chiefly devoted. It is only necessary further to say, or rather to repeat, that there are really two groups of cases included in the variety—viz., (1) that in which the disease is distinctly secondary to recurrent bronchitis or to chronic lung overstrain, and in which the degenerative changes supervene, and render the lesion permanent; (2) that in which the atrophic changes are primary, and the bronchitis and other phenomena are secondary. The latter cases are either hereditary or are induced by intemperate living, gout, syphilis, or other acquired cachexia.

2. *Small-Lunged or Senile Emphysema.*—This resembles the second group of the last variety in being an emphysema of essentially atrophic origin. It is, however, but the atrophy of old age most obvious at the lungs, but present everywhere. The pathology of this disease, if such it can fairly be called, is identical with that of the preceding, save that it supervenes in lungs already small and shrunken with the general atrophy of the body. Persons thus affected are prone to bronchial

attacks, and their heat-sustaining powers are very feeble; but under good conditions of warmth and clothing, with careful living, and a moderate amount of stimulants, they may enjoy life to even beyond the average age.

SYMPTOMATOLOGY.—The clinical phenomena presented by cases of marked emphysema are only in comparatively small part significant of the emphysematous lung, being largely due to attendant and for the most part secondary lesions. The physical signs will be sufficiently indicated in the description of the following extreme case of the disease:

George D——, oil and colour warehouseman, aged about fifty, was admitted into the Brompton Hospital under the care of Dr. Douglas Powell. The patient was a tall man, with no hereditary tendency to phthisis, but of gouty parentage on the father's side. He had up to three years before admission never been laid up with any illness. At that time he had an attack of bronchitis, and since then had complained of shortness of breath and cough, constant in the winter, attended with frothy and viscid expectoration and with a sense of constriction below the ribs. He had never spat blood nor suffered from hectic, but had lost and then regained flesh rapidly.

His principal symptoms on admission were great shortness and difficulty of breathing, the head and face becoming congested, almost cyanosed at times, during attacks of dyspnœa and cough. He had no pain, but the sense of constriction below the ribs, already mentioned, was marked. His appetite was bad, digestion tolerably good, bowels irregular, sleep fair.

Physical Signs.—Pulse 86. Temperature normal. Great œdema of lower extremities and scrotum. Chest greatly expanded. Respiration slow and forced. Respiratory movements mainly thoracic, and extraordinary muscles of respiration prominently employed. Intercostal spaces above the nipple level slightly depressed during inspiration, becoming quite level with the ribs on expiration. Below the nipple level the intercostals were greatly depressed during inspiration, becoming level with the ribs or even slightly puffed outwards during expiration. The seventh and eighth ribs yielded inwards with inspiration. The heart's impulse was most perceptible at left costal margin at the level of the tip of the ensiform cartilage.

Girth of chest on each side above nipple level, 18 inches; at the level of base of ensiform cartilage on the right side, $18\frac{5}{8}$ inches, and on the left side, $18\frac{1}{2}$ inches, with $\frac{1}{4}$ inch extreme inspiratory expansion. The whole chest was resonant down to the margin of the ribs both in front and behind. At the posterior bases on both sides fine bubbling râles were heard, principally with inspiration. Similar râles were also heard over the lower two-thirds of the right and left fronts. Apices clear. Cardiac sounds unattended with

bruit. Abdomen somewhat distended, and containing a small quantity of fluid. Liver depressed.

The subjoined tracings show well the nature of the respiratory movements in this case. They were taken by a simple apparatus, consisting of a straight rod connected by a flexible joint with an expanded button to apply to the chest, the other end writing upon a horizontal sphygmograph plate previously smoked.

Tracing Fig. 27 represents the movement of the sternum at the level of the third cartilage, the patient sitting in a chair, with his back

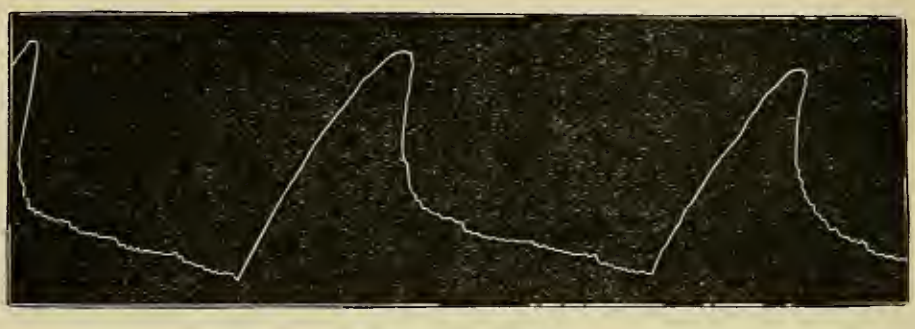

FIG. 27.—TRACING OF RESPIRATORY MOVEMENTS IN EMPHYSEMA, SHOWING TOTAL FORWARD THRUST AT THIRD MID-STERNUM.

resting against a flat board. It is equivalent to exaggerated thoracic breathing, although the patient was inspiring in the degree natural and necessary for him. Tracing Fig. 28 was taken on the seventh rib in the lateral region right side, and shows a distinct recession (*c, c, c*) during each inspiration. This was, perhaps, due to the bases of the lungs being in some measure disabled by secretion, but the disablement and collection of secretion were undoubtedly in greatest measure owing to the inaction of the diaphragm in consequence of the flattening of its arch. The thoracic movement was certainly

FIG. 28.—TRACING SHOWING RECESSION IN EMPHYSEMA (*c, c, c*) DURING INSPIRATION, SEVENTH RIB, AXILLARY LINE.

somewhat in excess in this case from the same cause, but in most cases of emphysema the respiration becomes thoracic rather than abdominal.

The patient died, after he had been in the hospital three weeks, from general dropsy and cyanosis.

The post-mortem examination revealed the usual phenomena of large dilated heart, with right side most affected, the tricuspid orifice measuring 6½ inches in circumference; large emphysematous lungs,

the emphysema most marked at the anterior and upper parts and in the right lung, together with œdema and slight congestion at both bases. The bronchial tubes contained a frothy thin fluid, but there were no signs of active bronchitis. The spleen was hardened, the kidneys mechanically congested, and the liver enlarged and fatty.

Treatment.—It will be obvious from what has preceded that the treatment of emphysema is mainly palliative, consisting of the prevention of fresh catarrhs, asthma, and bronchitis, the avoidance of over-exertion and straining occupations, the escape from dusty, irritating atmospheres, and, when possible, the timely migration to more genial climates during the winter or early spring months.

We cannot cure the disease, but we may by judicious measures arrest textural decay, and prevent fresh overstrain. Emphysema, let us again remark, is never fatal, within the normal period of human life, save by its complications, but it is the factor which endangers recovery from many diseases.

In the *dietetic* treatment we must so far restrict ingoings as to adapt the resulting products to (1) the needs of a necessarily limited muscular activity and a diminished metabolism; (2) to a somewhat retarded circulation through the lungs, the result of capillary obliteration; (3) to a lessened oxygenation and a corresponding tendency to plethora of venous blood.

A restricted, well-assorted dietary, and the maintenance in fair activity of the eliminatory functions of the skin, kidneys, and bowels, will fulfil the double indication of avoiding surcharge of the economy by waste materials and diminishing the tendency to venous plethora, visceral congestion and overwork of heart.

With regard to the treatment of emphysema by *drugs*, there are certain clear indications to be followed.

Measures for the regulation of secretions have been alluded to, and are of great importance, but require no further detailed description.

Intercurrent attacks of bronchitis or asthma will require appropriate treatment, but except at such times one should, so to speak, forget the lungs in the medicinal treatment of this complaint. Dyspnœa is not always to be regarded as an indication for ether nor a bronchial wheeze for squills. We must rather have careful regard to the general condition of the patient, and especially to vessel and heart tonicity. Iron,

arsenic, and strychnia are the best general tonics, and should be given in small doses for lengthened periods, say for a month at a time, with intervals of rest from drugs. The arseniate of iron is an excellent preparation for our purpose, *e.g.*, in $\frac{1}{12}$ to $\frac{1}{8}$ gr. doses, with $\frac{1}{2}$ gr. of extract of nux vomica and some pepsin, twice a day after food. A little aloes may be added if necessary, or an occasional morning dose of aperient waters advised, with more rarely a mercurial, with the view of maintaining equilibrium in the portal system. Ten drop doses of tincture of perchloride of iron, with a little strychnia, twice a day, will often prove of value in restoring muscular tone to the heart and to the bronchi, after any fresh attack of bronchitis or asthma involving renewed strain upon the right ventricle. Occasionally at these times it is desirable to give digitalis in moderate doses for a few weeks; five minims three times in the day is usually quite sufficient for the purpose.

In the advanced stages of emphysema and its concomitant affections, when the limbs become dropsical, the abdomen full, the viscera engorged, and—what is the key to the whole situation—with the right ventricle fluttering at the epigastrium, the pulse small, irregular, and intermitting, and the jugulars distended and filling from below, absolute rest in bed, the free administration of digitalis, with diuretics and diffusible stimulants, will sometimes still serve to rescue such patients from impending death. The flow of urine freely returns, the pulse steadies, and the dropsy subsides under this treatment. Albuminuria, usually more or less present under these conditions, is no contra-indication to the use of blue pill, which, given in combination with squill and digitalis for three or four successive nights, will frequently give a start in the direction of improvement.

The heart is the failing link in the phenomena present, and digitalis is the remedy, but it sometimes taxes our ingenuity and resolution to give the drug in a combination in which it can be borne. The digitoxin preparations, such as Nativelle's digitaline and Hoffmann's digalen, are sometimes usefully substituted for the more Galenic preparations. Some patients may take strophanthus better than digitalis, and the changes may be rung upon these two drugs. Convallaria may be tried as an alternative, but is by no means equal to either of the preceding in influencing heart and vessels. In the employ-

ment of these medicines pains should be taken to specify such preparations as are standardised and reliable.

Aerotherapeutics.—Much has been written and many observations made upon the treatment of emphysema and allied complaints by means of compressed and rarefied air. The idea is no new one, having been advocated by Nathaniel Henshaw in the seventeenth century in his interesting little work entitled "Aero-Chalinos; or, A Register for the Air,"[12] but it is chiefly to Waldenburg[13] that we are indebted for the modern employment of the method. Theoretically, expiration into rarefied air would appear in emphysema the more hopeful method, the rarefaction tending to extract the air from the distended lungs, and thus render easier the succeeding inspiration. In practice, however, better results are obtained from the use of compressed air. The treatment may be carried out in two ways: the patient may use a portable apparatus, of which Waldenburg's modification of Hutchinson's spirometer is the best known,[14] and by means of an appropriate adjustment secure the due compression or rarefaction of the air to be respired. Experience has, however, hardly borne out the hopeful results at first claimed from the use of such instruments, and as the continual adjustment of the necessary mouthpiece is often unpleasant to the patient, the method is now but little used.

More satisfactory results are obtained by the use of the compressed-air bath, in which the whole body of the patient, and not merely the surface of his respiratory tract, is subjected to the influence of the compressed air. Since the early experiments of Tabarié the method has been gradually improved, until the present satisfactory chambers have been devised. These may be seen at numerous places on the Continent, and notably at Reichenhall, in Bavaria. A detailed description of the one installed at the Brompton Hospital will be found in Dr. Theodore Williams's work on "Aerotherapeutics."[14a] It consists essentially of a chamber made of wrought iron $\frac{1}{16}$ inch thick, provided with iron door and plate-glass windows, sufficiently strong to resist the pressure to which the whole will be subjected, and containing chairs and tables for the convenience of the patients. The chamber is provided with a pump for compressing the air (worked in this case by steam), and a receiver containing cotton-wool, through which the air

is filtered. It may be made of any size; that at the Brompton Hospital will accommodate three or four patients, whilst others have been constructed to hold twenty.

The patient takes his seat in the chamber, prepared for a stay of two hours. The pressure of the air is then very gradually raised, until at the end of half an hour it exceeds the normal by ten pounds, or two-thirds of an atmosphere. At this point it is allowed to remain for an hour, and is then gradually reduced, until at the end of the second hour the normal has again been reached. If care be not taken to alter the pressure very gradually, various unpleasant sensations referable to the throat and membrana tympani will be experienced; but no danger of caisson disease need be anticipated, since to produce the latter affection it has been shown that a reaction from a pressure exceeding three atmospheres is necessary.

The baths should be given every other day at first, and then every day, a course lasting from six weeks to two months, after which they may be continued at longer intervals.

As a result of this treatment, there can be no doubt that certain cases of emphysema do improve considerably. The cough and expectoration lessen, the chest diminishes in circumference, and cardiac and liver dulness tend to return to the normal; the breathing at the same time improves, so that the patient is able without distress to take exercise, which before would have been impossible. How exactly these changes are brought about is not quite clear; it may be that they result from a beneficial action of the compressed air upon the bronchi, leading to a constriction of their bloodvessels and a diminution of secretion and spasm, rather than from any direct action upon the alveoli themselves.

Favourable results, such as we have described, must not, however, be looked for in every case; nor can it be predicted from the symptoms or physical signs what the result will be in any given instance. Although the method of treatment is empirical, it is one, nevertheless, which may be tried if the requisite facilities are available.

Climatic Treatment.—It is generally held that elevated climates are unsuitable for patients with emphysema, inasmuch as such patients must breathe more deeply to obtain the same amount of oxygen from a rarefied atmosphere. From

the following considerations, however, it would appear that such an atmosphere is much less unsuitable to emphysematous patients than one might at first assume. In the first place, it must be remembered that with an abundant air-supply only a very small proportion of the oxygen is used for respiratory purposes; in other words, expired air is not nearly exhausted of its oxygen. Secondly, although the air of elevated regions is rarefied, its particles are more actively mobile, and oxygenation is relatively quickened; thus, Tyndall and Frankland[15] demonstrated that the loss of weight of a candle burning on Mont Blanc at an elevation of 12,000 feet is identically the same as that of another candle of similar dimensions burning in the valley of Chamounix below. Thirdly, the circulation through the lungs, as elsewhere, is carried on at less pressure in elevated regions, and the heart, tuned originally to lower latitudes, finds relief in this way.

The above considerations are interesting, because they show that on theoretical grounds a certain amount of emphysema need be no bar to residence in a higher altitude if this be otherwise indicated. And this agrees with our practical experience, for we have known not a few cases of chronic phthisis with varying degrees of emphysema to do well in the High Alps. Nevertheless, we should not advise such climates for the more usual cases of chronic bronchitis and emphysema. For these, a warm climate near the sea-level is to be recommended, where the patient may take sufficient exercise without the exertion of hill-climbing, and where the danger of recurrent attacks of bronchitis is diminished as far as possible. Such conditions are to be found at many of our own health resorts, such as Falmouth, Ventnor, or St. Leonards; or abroad at such stations as Mentone, Bordighera, or San Remo, on the Riviera. In Egypt, Grand Canary, or Madeira, the conditions are also favourable.

Surgery.—During the last few years an attempt has been made, notably by Professor Delbet, of Paris, to deal surgically with emphysema[16] by the performance of *chondrectomy*—that is to say, the excision of certain rib cartilages with their perichondrium, on the assumption that loss of elasticity of the framework of the chest referred to by Freund (see p. 274) is the most important element in the malady. In certain cases relief has been given, but the operation is not free from

danger, and we believe that the cases are rare in which such treatment can justifiably be entertained.

Interstitial or Interlobular Emphysema.

This condition depends on the escape of air into the connective tissue surrounding the lobules, bronchioles, bronchi, and bloodvessels, and also that beneath the pleura. It is caused by wounds of the lung tissue, or by rupture of the air-vesicles during severe attacks of cough or other straining efforts. It is most common in childhood during the paroxysms of whooping-cough and other diseases associated with violent and prolonged coughing, but we have known it to occur in the course of phthisis.

If limited to the lung, the air appears as small beads in the interlobular tissue; but under the pleura it may form blebs of considerable size (Plate XV.). It further tends to pass into the mediastinum, and thence into the cellular tissue of the neck, producing surgical emphysema, whence it may even extend over the trunk. In rare cases it has led to pneumothorax. If left alone the air is usually absorbed, but if there is great tension this may be relieved by puncture or incision of the skin, when the air escapes.

If uncomplicated and limited to the lung, the condition gives rise to no clinical symptoms or physical signs, and is only discovered at the autopsy.

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(*b*) *Loc cit.,* p. 7.

[2] (1) "The Mechanism of Respiration in Man," by Arthur Keith, M.D. (with bibliography), being an article in *Further Advances in Physiology,* edited by Leonard Hill, M.B., F.R.S., p. 182. London, 1907.
(2) "Why does Phthisis attack the Apex of the Lung?" by Arthur Keith, M.D., *The London Hospital Gazette,* January, 1904.

[3] See article on "Emphysema and Atelectasis," by Prof. Friedrich A. Hoffmann, in Nothnagel's *Encyclopedia of Practical Medicine* (English edition), edited by John H. Musser, M.D., pp. 258-263. Philadelphia and London, 1903.

[4] "On the Determining Causes of Vesicular Emphysema of the Lung," by William Jenner, M.D., F.R.C.P., *Transactions of the Royal Medical and Chirurgical Society,* 1857, vol. xl., p. 25.

PLATE XV

INTERSTITIAL EMPHYSEMA

On the surface of the lung numerous air-bubbles are seen, from the rupture of alveoli and the escape of air into the tissues beneath the pleura.

From a male child aged nine and a half months, who died from whooping-cough. The section of the lung showed patches of broncho-pneumonia, but no vesicular emphysema.

(From the Museum of St. Bartholomew's Hospital.
Natural size.)

PLATE XV

INTERSTITIAL EMPHYSEMA.

To face p. 284.

[5] *Traité de l'Auscultation Médiate et des Maladies des Poumons et du Cœur,* par R. T. H. Laennec, troisième édition, tome i., p. 292. Paris, 1831.

[6] *On the Pathological Anatomy of Bronchitis, and the Diseases of the Lung connected with Bronchial Obstruction,* by W. T. Gairdner, M.D., p. 57, etc. Edinburgh, 1850.

[7] *Medical Lectures and Aphorisms,* by Samuel Gee, M.D., fourth edition, p. 127. London, 1915.

[8] *On Bronchitis and the Morbid Conditions connected with it,* by Edward Headlam Greenhow, M.D., F.R.S., second edition, pp. 233-237. London, 1878.

[9] *Der Zusammenhang gewisser Lungenkrankheiten mit primären Rippenknorpel-Anomalieen,* von Dr. Wilhelm Alexander Freund (zu Breslau). Erlangen, 1859.

[10] *Die Pneumatometrie und Spirometrie,* von Dr. L. Waldenburg, p. 56, 1880.

[11] See cases recorded by (*a*) the late Professor Coats, *Manual of Pathology,* by Joseph Coats, M.D., p. 739 (London, 1895); also (*b*) by Dr. Wethered, "A Case of Hypertrophy of the Lung," by Frank J. Wethered, M.D., *Transactions of the Pathological Society of London,* 1897, vol. xlviii., p. 34.

[12] *Aero-Chalinos: or, A Register for the Air,* by Nathaniel Henshaw, M.D., F.R.S., the second edition. Printed for Benj. Tooke at the Ship in St. Paul's Churchyard, London, 1677.

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[14] See (*a*) *Aerotherapeutics,* by Charles Theodore Williams, M.D., p. 98. London, 1894.
(*b*) "Artificial Aerotherapeutics," by C. Theodore Williams, M.D., Allbutt and Rolleston's *System of Medicine,* vol. v., p. 35. London, 1909.

[15] See *On the Curative Effects of Baths and Waters,* by Dr. Julius Braun, edited by Hermann Weber, M.D., p. 59. London, 1875.

[16] See (*a*) "Freund's Operation in Pulmonary Emphysema," *British Medical Journal,* January 8, 1910, epitome No. 20, p. 6.
(*b*) "Le Thorax et l'Emphysème : la Chondrectomie," par E. Douay, *Annales de la Clinique Chirurgicale du Professeur Pierre Delbet.* Paris, 1914.
(*c*) "Operation for Emphysema," *British Medical Journal,* 1916, vol. ii., p. 428.
(*d*) *Surgery of the Lung and Pleura,* by H. Morriston Davies, M.A., M.D., M.C., F.R.C.S., p. 246.

CHAPTER XVII

PNEUMONIA

ACUTE lobar pneumonia, or, as it is inaptly termed by German writers, "croupous pneumonia," may be defined as an acute specific disease, characterised by inflammatory consolidation of some portion of one or both lungs.

In a sense, pneumonia and inflammation of the lungs are synonymous terms, but the latter expression by no means covers the whole pathology of the disease. In many, if not in all cases the micro-organisms responsible for the malady pass into the blood-stream, and under certain conditions specific complications in distant organs, such as the meninges of the brain or the internal ear, result. In all cases, too, the poisons elaborated in the lungs are rapidly absorbed, and to their toxic effects many of the most characteristic symptoms are due. In every case, therefore, the disease affects the whole system to a greater or less extent, and any conception which regards it as limited to the lungs must be erroneous. We have to deal, in fact, in pneumonia, with a blood infection as well as a local inflammation.

Ætiology—*Individual Predisposition.*—No age is exempt from pneumonia, but the malady is more prevalent at that period between twenty and forty, when persons are most exposed in the active struggle of life. The male sex is more frequently attacked than the female in proportion of three to two, and at the period of life referred to the prevalence is twice as great amongst males (Longstaff[1]). Depressed vitality, arising from debauchery, intemperance, over-fatigue, anxiety, insufficient food, or overcrowding, renders the individual more prone to attack. Some persons are especially liable to the disease, and one attack favours the disposition to future recurrences. In rare cases as many as thirteen, fifteen, sixteen, or even twenty-eight attacks have been recorded.[2a]

Pre-existing Diseases.—Chronic disease of any kind, but

especially alcoholism, albuminuria, and gout, increases the liability to pneumonia. But, curiously enough, the presence of already existing chronic lung affections, such as phthisis, asthma or bronchitis, does not appear to render the subject more liable to the disease. A plethoric state of body seems to favour its occurrence, and to add much to the severity of the attack. Surgical injury and the shock resulting from severe accidents are not infrequently followed by its appearance, especially in old people.

Climatic Influences.—Cold seasons, great variations of temperature and rough piercing winds bring about pneumonia. The disease is thus comparatively rare during the summer from June to October, but with the advent of winter its frequency increases, until its maximum incidence is reached in February or March, when in this country the east winds are most severely felt.

It is to be observed that a merely low external temperature is not so favourable to the occurrence of pneumonia as exposure to great variations. Indeed, it is stated that pneumonia is more common in hot than in cold climates. Chill from undue exposure to draughts, cold winds, and the like, is the most common exciting cause of pneumonia, and the disease not infrequently arises in workmen who, getting warm at their work in the sunshine of a May day, throw off coverings and become chilled by a north or north-east wind, of which they were before unconscious. Not only are most of the sporadic cases to be accounted for in this way, but a certain number of the epidemic occurrences of the disease in modern times are thus explained, individual susceptibility and special exposure being the predisposing circumstances. The epidemic related by Assistant-Surgeon Welch[3] as affecting the 22nd Regiment, stationed at New Brunswick, may be quoted as an example. The troops, who had been stationed at Malta for six years, were transferred during cold wintry weather to New Brunswick. Of the total strength of 652 men, 52 were soon attacked with pneumonia, 12 cases occurring in January, the month of greatest cold, and 31 in February and March, when temperature fluctuations were most marked. Of the 652 men, 330 were housed in the "Exhibition building," a large, cold, draughty wooden structure, freely exposed on all sides, and of these 38 (or 11·5 per

cent.) developed the disease. The remaining 322 were quartered in suitable and warmer barracks, or in married quarters, and of these only 13 (or 4 per cent.) were affected; whilst among 152 women and children only 2 cases occurred. Of the whole 52 cases, Welch attributed 27 to cold draughts and lowness of general temperature, 7 to sleeping on mattresses carelessly stuffed with damp snowed straw, 5 to exposure to great cold at night, and 6 to chill whilst perspiring from strong exercise. In the remaining 7 cases the direct exciting cause was obscure. The facts here recorded are striking, and emphasise the important part played by "exposure" in the production of the disease.

Injury.—In a small proportion of cases a blow on the chest has seemed to be the direct exciting cause of acute pneumonia, and such cases have been styled "traumatic" or "contusional" pneumonia.[4] But, apart from these, we have met with cases that have been apparently attributable to the shock following injury to another part of the body, and we may note that a similar phenomenon has been observed in the case of massive collapse of the lung (see p. 346).

Septic Influences.—Ætiological researches, and especially the evidence brought together by the Committee of Collective Investigation of the British Medical Association,[5] leave no room for doubt that cases and groups of cases of pneumonia are to be met with which are dependent upon faulty sanitary conditions, and especially exposure to sewer-gas emanations. Whenever several persons in a house are attacked, either together or in quick succession, the possibility of such a cause should at once suggest itself. The disease, when thus arising, is apt to be of a somewhat different type from the more ordinary variety, and merits the term "septic pneumonia," under which we shall describe it.

Direct Infection.—There is evidence to show that, under certain circumstances, pneumonia may be communicated directly by one person to another. The Collective Investigation Committee, in answer to a special request for information as to the ætiology of pneumonia, received about one hundred replies, in eighty of which no other cause than exposure to cold could be assigned for the attack. The twenty remaining replies are abstracted in the Record[6a] and amongst them nine observations are included in which there was apparent transmission

of the disease from one member of a family to others, in at least one of which the evidence is very strong.

The difficulty is, of course, the usual one of separating cases of several persons being attacked in consequence of exposure to a common cause from those in which the first attacked has transmitted the disease to others. In the epidemic at New Brunswick already referred to no suspicion of communication of the disease by infection is mentioned, and similarly with one of the two epidemics described by Surgeon-Major Maunsell[7] in North-West India. Several striking instances of the apparent origin of pneumonia by contagion are, however, quoted by Drs. Sturges and Coupland[8] from various authors, and others have since been recorded.[9] Some of these are convincing, as when a person has acquired the disease after nursing a sufferer from pneumonia, and, having been sent to a distant home, other cases have at once arisen among the members of the fresh family previously in perfect health. Cases such as these—and not a few have now been recorded—are difficult to put aside, and we must conclude that in certain circumstances, when the virus is especially active or the system more than usually receptive, the disease may be spread by direct infection. Indeed, it is not improbable that, were the sputum in pneumonia less viscid, and were it therefore easier for infected droplets to be coughed into the air, and thus inhaled, the disease would be more often communicated in this manner. In this connection it should be noted that the pneumococci responsible for the attack disappear from the mouth as a rule within three to four weeks after recovery, but they have been known to persist as long as three months.[10a]

Bacteriology.—Bacteriological research has demonstrated that in the great majority of cases of lobar pneumonia, perhaps 95 per cent., the pneumococcus discovered by Talamon and further studied by Fränkel is to be found in the lung, and often it is the only organism present. That it is capable of producing pneumonia is shown by the fact that if a culture be injected into the lung of a sufficiently resistant animal, such as the dog or sheep (Gamaléia[11]), acute lobar pneumonia, identical with that occurring in man, results. We are justified, therefore, in regarding the pneumococcus as the specific cause of the disease in the vast majority of cases.

In a small percentage of patients other organisms may be responsible, such as the pneumo-bacillus of Friedländer, which has occasionally been found in pure culture in the lung, or the pyogenic cocci which are sometimes alone present in so-called "septic pneumonia."

The preponderant rôle played by the pneumococcus in cases of acute lobar pneumonia is demonstrated by the following figures, which give the ætiological agents concerned in 480 cases of the disease treated at the hospital of the Rockefeller Institute in New York, in which the bacteriological findings were fully worked out:[10b]

Diplococcus pneumoniæ	454
Friedländer's bacillus	3
Pfeiffer's bacillus (of influenza)	6
Streptococcus pyogenes	7
Streptococcus mucosus	1
Staphylococcus aureus	3
Mixed infections, with combinations of Staphylococcus aureus, Friedländer's bacillus, Pfeiffer's bacillus, Streptococcus pyogenes, and Streptococcus viridans	6
Total	480

In the sputum or in films made from the pneumonic exudation the pneumococcus presents the appearance of small cocci, often arranged in pairs, the two ends of which are generally somewhat pointed. It thus acquires a lancet-shaped appearance, and has been accordingly termed the *Diplococcus lanceolatus*. The organism stains by Gram's method, and, when growing in the tissues, is surrounded by a definite and characteristic capsule. To demonstate this in the sputum, a portion should be dried on a cover-glass and stained with methylene blue, then washed and mounted in water. In this way, as the late Professor Kanthack showed, the capsule is easily brought into view. At a temperature of 37° C. cultures may be obtained without great difficulty, and on agar they present a characteristic appearance, forming small round transparent masses, which have been compared to drops of dew. Below 24° C. but little growth occurs.

Pneumococci are most easily demonstrated in those areas of the lung which are in the earliest stage of the pneumonic process and in a condition of acute inflammatory congestion. But they are not confined to the lung, and may be found in

the pleura or pericardium, when these tissues have become infected and inflamed by direct extension. Often also they are present in the blood, and in this way may be responsible for a malignant endocarditis, a meningitis, an otitis or arthritis, or some other and distant complication.

We have spoken hitherto as though the pneumococcus were a single organism of unvarying character, but recent researches at the Rockefeller Institute in New York have revealed that, just as in the case of the streptococcus and the colon bacillus, more than one type of pneumococcus may be recognised. Differentiation of the strains can be made by the agglutination test, using sera prepared by the inoculation of various strains of pneumococci, and in this way three types (I., II., and III.) may be easily separated. A fourth variety, which does not agglutinate with any of the standard sera, but which, like other pneumococci, is soluble in bile, has also been isolated. The following table, taken from the valuable monographs of the Rockefeller Institute to which we have referred, shows the frequency of occurrence in New York, as well as the percentage mortality, caused by each variety or type: [10c]

Incidence in New York of Various Types of Pneumococcus and resulting Mortality.

Type.	Incidence. Per Cent.	Mortality. Per Cent.
I.	33	25
II.	31	32
III.	12	45
IV.	24	16

We thus see that pneumococci of Types I. and II. are responsible for 64 per cent. of all cases of acute lobar pneumonia occurring in New York, and that the majority of deaths from this disease are due to their agency. Few cases of pneumonia are produced by organisms of Type III., but their virulence is great, and nearly half the patients attacked die. Type IV. is responsible for nearly a quarter of the cases of pneumonia, but the mortality is less, a fatal termination occurring in only 16 per cent. of those attacked. An identification of the type of pneumococcus present in any given case has thus an obvious bearing upon prognosis, as well as upon the question of specific treatment, to which we shall refer later. We may add that in the pneumonia which occurs so frequently in South Africa among the natives working in the

gold mines, Dr. F. S. Lister[12] has established other varieties of pneumococci, in addition to those discovered by the workers at the Rockefeller Institute, one of which he finds to be of frequent occurrence and of high case mortality.

In addition to being present in the tissues of patients suffering from pneumonia, the pneumococcus, as Pasteur[13] first showed, may be found in the saliva of not a few healthy people (Netter[2b]). The researches of the Rockefeller Institute would appear to indicate that such pneumococci belong for the most part to Type IV., the least virulent of the various strains, and that, except in convalescents and "contacts," organisms of Types I. and II., which are responsible for the majority of cases of pneumonia, are but rarely met with. Nevertheless, the fact that pneumococci do occur in the mouths of nearly 40 per cent. of normal persons[10d] is a fact which helps to elucidate a good deal that has hitherto appeared obscure in the ætiology of pneumonia, and it is not difficult to understand how cold, wasting diseases, or alcoholism may predispose or give rise to the disease, provided the specific germ is already present within the body, ready to take advantage of the local vascular changes or lowered resistance which favour its growth.

Pathology and Morbid Anatomy.—The morbid anatomy of pneumonia consists, in the first place, of an acute hyperæmia of the affected lung, resulting in a fibrino-corpuscular exudation into the alveoli, sometimes including the smaller bronchioles, and forming a film upon the pleural surface. This exudation, coagulating *in situ*, fixes the lung in a state of immovable expansion, more densely solidified than it could be by any artificial injection with coagulable fluid. In the attainment of this second stage of "hepatisation," all the severity of the disease is manifested, and on its completion the disease proper is at an end. Thirdly, with liquefaction and absorption of the morbid products, convalescence becomes established.

A normal pneumonia is thus artificially divisible into three stages.

The *first stage* begins with the rigor, and ends with the appearance of definite signs of consolidation. It may be termed the stage of initial fever with pulmonary engorgement, and it lasts from two to five days. If death should

occur at this stage, the affected lung is found to be in a condition of inflammatory œdema, heavy, engorged with florid blood, pitting on pressure, and still crepitating. On section, abundant blood-stained and frothy serum exudes. Pulmonary hyperæmia and fluid exudation are the conditions present. When the pneumonia occurs in very cachectic subjects, scattered pulmonary hæmorrhages may be found. A mere outburst from engorged vessels at some one time, however, is not necessarily associated with disease of a specially low

FIG. 29.—SECTION OF LUNG IN PNEUMONIA: LATTER PERIOD OF RED HEPATISATION.

type. It must be observed that throughout the febrile period of pneumonia there is an advancing area of lung thus affected.

The *second stage*—that of pulmonary hepatisation—emerges from the former, and terminates in from forty-eight hours to five or six days. It is characterised by continued high temperature, and by increasing signs of consolidation of lung. In this stage of *red hepatisation* the affected portion of lung is bulky, heavy, and solid to the feel. The pleural surface is more or less covered with a thin layer of soft, finely granular lymph which can be readily scraped off, exposing the glistening pleura beneath. In some cases this layer is complete, of some thickness, and of yellowish opacity. On sec-

tion, the lung is firm and dry, presenting a red granular surface, which is readily broken by the pressure of the finger. A little frothy secretion may be present in the bronchial tubes, the mucous membrane of which is injected. If the cut surface be scraped, and the juice thus obtained be examined, casts of the alveolar passages and bronchioles will be found. A portion cut from the consolidated lung sinks at once in water.

A thin section from the consolidation shows the alveoli, infundibula, alveolar passages, and sometimes the smallest divisions of the bronchi, to be occupied by red blood-discs and leucocytes entangled in the meshes of coagulated fibrin (Fig. 29). The alveolar wall is not changed beyond sometimes showing slight swelling of its epithelium, a few cells of which may be shed. Towards the latter portion of this stage there is more or less emigration of leucocytes through the alveolar walls. In cases in which the pulmonary inflammation supervenes upon long-continued hypostatic congestion the consolidation is softer, darker, and more spleen-like in appearance, and has been called "splenification of lung." The term "hypostatic pneumonia" is also applied to this condition.

Third Stage.—That of resolution. The commencement of this stage is characterised in normal cases by a remarkably rapid fall of temperature, attended with profuse sweatings or other critical phenomena, and the signs of commencing liquefaction of the elements of the lung consolidation. The lung now assumes a greyish aspect, with some red patches still remaining. On section, the granular appearance is at first maintained. Later, the whole lobe becomes of a dirty greyish-yellow colour and much softer. This stage of pneumonia is spoken of as *grey hepatisation.*

The microscopical appearances are at first characterised at this stage by an extensive emigration of leucocytes. The exuded products then undergo degeneration, the fibrinous strands becoming completely disintegrated, the red corpuscles decolourised, and finally both red and white cells more or less broken down by fatty changes. The change in colour observed on the post-mortem table from red to grey hepatisation is brought about partly by these alterations, partly through compression of the pulmonary capillaries by the pressure of the exudation.

As a rule, the alveolar walls remain intact, beyond, perhaps, manifesting slight swelling of their epithelial lining, and the emulsified products are readily removed, chiefly by absorption, but in part also by expectoration. In some cases, however, the texture of the lung is involved in the inflammatory change, the alveolar walls become infiltrated with leucocytes, and the reparative stage of grey hepatisation is changed for that of suppurative disintegration of the lung itself. This untoward event, spoken of as "*purulent infiltration*," must be regarded as a complication rather than as a feature of pneumonia. It is doubtful whether recovery can take place when this condition has been established. Should it occur over a localised area, an abscess of the lung results.

The portion of lung affected in pneumonia is most commonly the base on one side, the right being more frequently affected than the left. It is rare for both bases to be simultaneously attacked, but it is common for the second base to become involved in the course of the disease. Perhaps the frequency with which this happens has been exaggerated from the fact that both tubular breath-sound and crepitant râle may be audible at one base by reflection from a corresponding point of the other. The upper lobe is attacked in a goodly proportion of cases, more commonly so in children than adults; amongst the latter are included the more cachectic cases, whether from alcohol or other causes, which generally run a graver course.

It must be borne in mind that the above description of the three stages of pneumonia relates to the pathology of each portion of lung attacked, but pneumonia frequently attacks successive portions of lung, when the morbid anatomy and symptoms of the three stages will be confused and overlapping.

It must be further noted that, apart from definite complications, a certain degree of cloudy swelling of the kidneys, liver, and heart is commonly to be observed, as in other specific fevers. Some swelling of Peyer's patches is also occasionally noticed.

Symptomatology.—The invasion of pneumonia is generally sudden, and is usually marked by a distinct rigor. Often, however, the attack is preceded by a few days of what may be regarded as prodromal symptoms, malaise, bronchial catarrh, anæmia, coated tongue, and slightly icteric tint of

skin. It is not uncommon for patients to have been out of sorts, depressed, dyspeptic, catarrhal, with torpid liver functions and loaded urine, for some weeks previous to an attack of pneumonia; but this is only another way of saying that they have been qualifying for an acute illness, the exact nature of which is determined by other circumstances, and which, by timely attention to the general health, might have been altogether avoided.

In place of the rigor which ushers in pneumonia, there may be, especially in children, convulsions or vomiting. The temperature rises with great rapidity from the time of the rigor; severe headache, and even somewhat violent delirium, may be present; the eye is as a rule bright, conjunctivæ sometimes icteric, face notably flushed, breathing hurried and regular, and attended with action of nares. The pulse is from the first always quickened in pneumonia, and especially so in young people, but not proportionately to the breathing. It must be recognised, however, that temperament and habitual pulse frequency have to be taken into account in estimating this symptom. There may be severe gastric disturbance, with slight jaundice, in the early days of the disease. Of local symptoms, pain and dyspnœa are those which chiefly attract attention.

The general symptoms often precede the appearance of physical signs by a very perceptible interval of time, even five or six days, during which, on examining the chest, no dulness is to be found. The breath-sounds at one base have, however, at a very early period a peculiar rough, harsh quality, very like that of exaggerated breathing. A few hours later the characteristic fine hair crepitation may be abundantly evident, whilst the percussion note, although shortened and heightened in pitch, is as yet by no means dull. In other cases the physical signs of complete consolidation may develope so rapidly as to render any previous stage unnoticeable.

Description of a Case.—For convenience of description of the general aspect and physical signs of pneumonia, we will commence with the second or third day after the rigor, the time at which the practitioner is most commonly called upon to see such a case, and at which as a rule there is not much room for doubt as to its nature.

The flushed look and burning skin; the hurried, noiseless breathing; the rapid but regular pulse; the frequent short cough, half-stifled from pain; the dryish thickly coated tongue; and the singular prostration of the patient form a group of signs which, supervening speedily upon a well-marked fit of shivering, cannot be otherwise interpreted than as characteristic of an attack of ordinary acute basic pneumonia in full intensity. A crop of herpes will often be observed upon the lip.

On closer examination the respirations are found to number about forty in the minute. They are not obstructed, but are usually attended with well-marked action of the nares. The pulse is 120, the temperature 104° (Fig. 30). On inspecting the chest, its movements are seen to be chiefly one-sided; but there is no apparent difference in size on the two sides, and the heart is found beating in its normal position. The movements of the affected side are voluntarily restrained by the patient, and a severe pain referred to this part often cuts short the cough or any attempt to draw a full breath. The percussion signs in front are not materially altered. On auscultation, the respiratory murmur is found to be weaker on the affected, exaggerated on the healthy side. Posteriorly over the base of the diseased lung the percussion note is dull, but not without some wooden quality of tone quite distinct from the dead flat note of effusion. The dulness extends upwards to a variable height, and over the dull portion the vocal fremitus is increased, the respiration is characteristically bronchial or tubular, the voice-sounds well conducted and bronchophonic, whilst the peculiarly explosive fine inspiratory crepitation of pneumonia is heard, especially towards the upper limits of dulness. If there be any expectoration, it is scanty, viscid, frothy, and more or less rust-coloured from blood-staining. It contains an excess of chlorides, and is deficient in phosphates. The urine is scanty and concentrated, and as in certain other febrile states, notably typhus and typhoid fever, is very deficient in chlorides, owing to their increased retention in the body.[14] A similar condition has been noted in acute phthisis. In about half the cases the urine yields a thin cloud of albumin, and in a small percentage definite nephritis, with blood-casts, is observed.

The blood shows peculiar changes. Its clotting power is

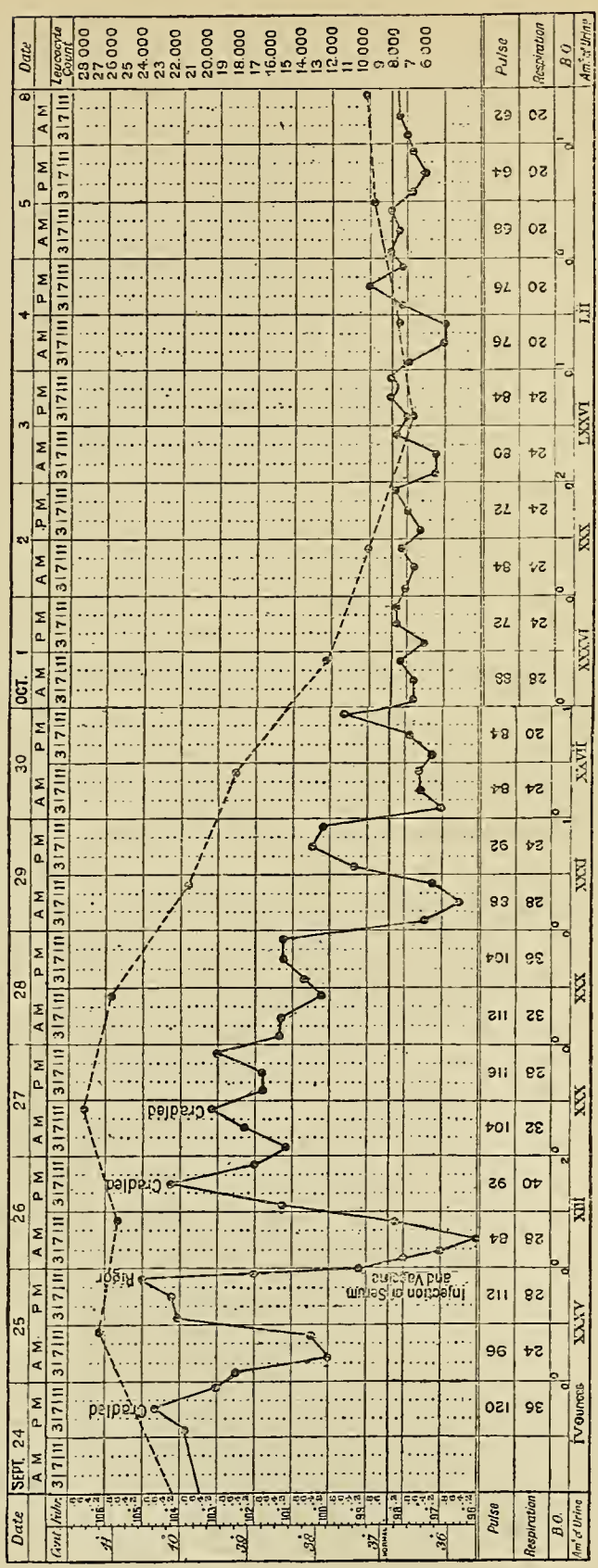

FIG. 30.

Chart showing the temperature record and leucocyte count (dotted line) of a case of pneumonia occurring in a man aged 63, who was admitted into St. Bartholomew's Hospital on the fourth day of the disease.

increased, and the amount of fibrin formed is greater than usual. These facts were well known to the older writers, who regarded the rapid formation of a firm clot after bleeding as of good prognostic augury. No doubt, too, the peculiar characters of the pneumonic exudation, and the presence of the fibrin network, are dependent on these changes. In most cases of pneumonia a considerable increase in the number of white corpuscles is also to be observed, the total per cubic millimetre commonly reaching 20,000, and sometimes even 50,000 or more. The increase begins early in the disease, and gradually becomes more marked, until with the crisis the number again quickly falls to normal, although not, as a rule, quite so rapidly as the temperature (see Fig. 30). This leucocytosis, as we shall see, possesses some diagnostic importance, and its absence in an undoubted case of severe pneumonia points to a lack of reactive power on the part of the patient, and warrants a grave prognosis. In many cases, as we have seen, the pneumococcus may be detected in the blood, and from the observations made at the hospital of the Rockefeller Institute, where 448 patients were examined, it would appear that this occurs chiefly in the graver cases and those with a higher mortality.[10e]

To follow our assumed case of pneumonia further onwards; the temperature, rising after the onset with rigor to 104°, hovers thereabouts, with but little variation (unless influenced by treatment) for four, five, six, or seven days, and occasionally longer, and then rapidly falls within thirty-six hours to normal. This abrupt fall of temperature, which marks the crisis, is often attended with profuse sweats, a copious discharge of urine depositing abundant lithates, and more rarely with diarrhœa. Some exhaustion is always present, and at times there is decided collapse. After this brief period of shock has passed, the patient, who has been suffering throughout from complete anorexia, thirst, restlessness, and increasing weakness, with dyspnœa, troublesome cough, and blood-stained expectoration, is rapidly relieved from many of these symptoms. The sense of dyspnœa is greatly lessened, although the breathing is still accelerated. The pulse becomes quiet, the skin moist, and the tongue begins to clean at the tip and edges. Appetite does not yet return, but sleep is quiet and refreshing. Such a crisis occurs in about sixty per cent. of all cases; in others the tem-

perature drops more slowly by lysis, and the amelioration of symptoms is correspondingly more gradual. In others, again, the crisis is overlapped by a renewed attack on the opposite side, with its attendant return of fever.

On examination after the fall of temperature the physical signs will be found, contrary perhaps to expectation, to have but little changed. Dulness is still present, and bronchial breathing and bronchophony are as distinct as ever. Soon, however, crepitant sounds somewhat resembling the fine-hair crepitation heard at the commencement of the disease, again make their appearance. These are, however, larger, moister, less explosive, and are heard during expiration as well as inspiration, although still most abundantly with inspiration. This subcrepitant râle, or *redux crepitation*, as it is termed, marks the commencing resolution of the hepatised lung, and extends downwards as the consolidation slowly melts away. The sputum, usually still scanty, now becomes more opaque; in certain cases it is muco-purulent and amounts to several ounces during the day. In no case is lung tissue to be discovered in it. Sometimes even in the adult, and often in children, there is from first to last no expectoration in pneumonia.

After the period of crisis there is in some cases a slight return of fever for a few days, but of a character different from that of the original disease. The temperature assumes a hectic type, with a moderate daily rise to 101° or even 102°, and in these cases slight daily chills may be experienced. These symptoms are somewhat alarming, and suggest the possibility of some complication. But they do not necessarily bear this interpretation, for we have observed them in several cases in which recovery has been otherwise uneventful. The explanation is possibly to be found in the too rapid absorption of inflammatory products, the toxic quality of which has not as yet been entirely neutralised.[15]

Diagnosis.—In a characteristic case of acute lobar pneumonia the diagnosis is not as a rule difficult. The sudden onset, the character of the temperature chart, the hurried breathing, with working alæ nasi, should at once suggest the disease. Later the rusty sputum and development of physical signs render the case clear.

But in other instances, as in the septic variety, which we shall

presently describe, the localising signs are less definite, and for a time it may be difficult to decide between typhoid fever and pneumonia. In such circumstances the blood should be examined, a definite leucocytosis, if found, speaking strongly in favour of pneumonia, a positive Widal reaction, on the other hand, deciding in favour of typhoid fever.

In other cases the diagnosis from pleural effusion is not altogether easy. Thus we have observed more than once well-marked examples of dense consolidation, involving the whole lung, in which breath-sounds were inaudible and vocal fremitus correspondingly diminished. The diagnosis of pneumonia was arrived at principally from the very slight cardiac displacement, which would have been far more extensive had the condition been an effusion into the pleura. In the cases referred to, redux crepitation subsequently developed and the consolidation cleared up. Doubtless the absence of breath-sounds was due to an unusually large overflow of coagulating exudation into the smaller bronchi. Similar cases have been recorded by other observers,[16] in which at the autopsy this explanation has proved to be correct. Again, as we have pointed out in dealing with pleurisy, loud bronchial breathing may be sometimes heard over an effusion, and suggests consolidation of the lung. Here, also, evidence of displacement or otherwise of the heart is of great importance, and should indicate a right diagnosis. In any case in which there is real doubt the exploring syringe should be used.

But beyond the diagnosis of inflammation of the lung, we have further to look, in view of treatment and prognosis, to the conditions which have led up to, caused, or which complicate the attack. Has the disease supervened upon shock or exhaustion, alcoholism or uræmia? Is it the result of some poison influence or sewer-gas emanation, to which, it may be, the patient is still exposed? Has exposure to severe chill or direct cold resulted in inflammatory congestion or consolidation in a strong, full-blooded, or a delicate person? The questions thus formulated can only be answered by separate inquiries in each case, but they are of even more importance than a merely physical diagnosis.

Varieties of Pneumonia.

The following varieties of pneumonia, which differ somewhat from the ordinary type, may now be described.

Septic Pneumonia.—This variety, formerly spoken of as *typho-* or *pythogenic pneumonia*,* is marked by a less sudden onset, greater prostration, a more prolonged course, and by less pronounced pulmonary symptoms than are usually observed. It, therefore, not uncommonly suggests typhoid fever. The following is a fairly typical example of this form of the disease:

Minnie L., aged nineteen, a nursemaid living in London, a light-haired, well-nourished girl, was admitted into the Middlesex Hospital, under Dr. Douglas Powell, on November 27, 1882. She had a family history of chest delicacy, but had herself suffered from no previous illnesses. Several persons in the house where she resided were stated to have complained recently of feverish and abdominal symptoms, and the water of which the patient had drunk freely was regarded with suspicion, and directions had been given that it should be boiled before being used for drinking purposes.

The patient had returned to town from Folkestone at the end of October, and had been in her usual health, except for slight headache during a fortnight, until November 23, when she was taken ill with shivering, frontal headache, pains in the back and limbs, giddiness, nausea, and cough, without expectoration. The shivering was repeated several times during the interval between the date of her attack and her admission to the hospital, the fourth day of illness. The skin was then found to be hot and dry, the lips parched and cracked, the tongue coated, and the bowels confined. No physical signs could be discovered except some enlargement of the spleen. On the eighth day imperfect dulness and patchy crepitations were observed at the left base, with bronchial breathing. On the tenth day some tubular breath-sound was also heard at the left subclavicular region. On the thirteenth day there was patchy dulness and crepitation over the left back and front of the chest, exaggerated breathing in the right subclavicular region, and scattered crepitation over the right axillary region and posterior base; rusty sputa; delirium.

On the fourteenth day the note was as follows: *Right side:* Considerable resonance of tubular quality over tenth and eleventh ribs posteriorly, with dulness above, including the region of the scapula. Coarse râles at the extreme base; inspiratory crepitation over scapula. In front exaggerated respiration; in lower axilla subcrepitant râles.

* The term "pythogenic" was originally suggested by the late Dr. Charles Murchison[17] for Typhoid Fever, and was first applied to pneumonia by Drs. Grimshaw and Moore.[18]

Left side: Posteriorly, patchy dulness, with coarse scattered liquid râles. Anteriorly, dulness in subclavicular region, with moist râles and some larger clicks. One doubtful spot suggestive of typhoid fever was observed on the abdomen. Death occurred on the morning of the sixteenth day. The appended chart (Fig. 31) gives the temperature of this case, and illustrates fairly well the more prolonged and fluctuating course of the fever which differentiates typical cases of this variety from those of the more ordinary form of pneumonia.

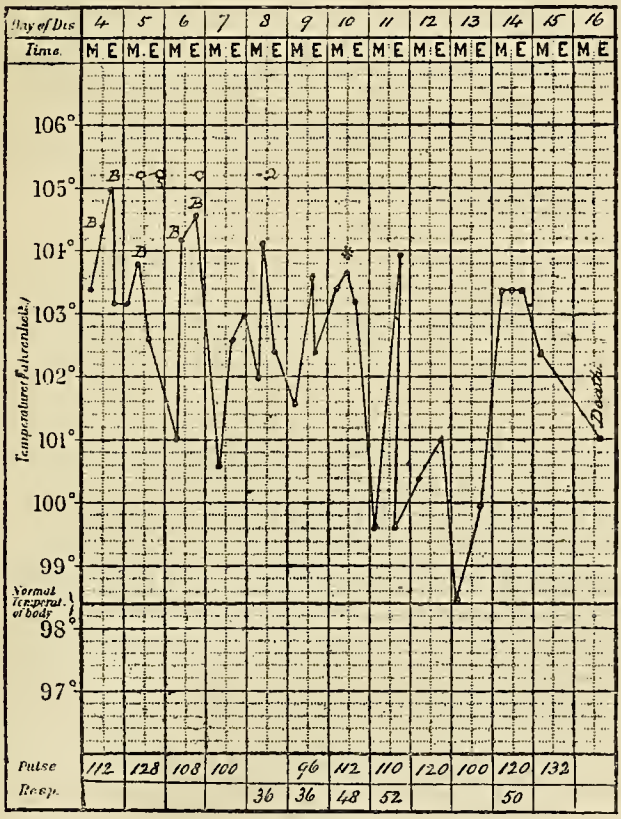

FIG. 31.—CHART SHOWING THE TEMPERATURE RECORD OF M. L., AGED NINETEEN, WHO SUFFERED FROM SEPTIC PNEUMONIA.

B = cool bath; Q = quinine (10 grains); * = salicylate of soda (20 grains every four hours).

Post-Mortem: no lesions of typhoid fever were found.

The upper lobe of the right lung was solid, except at its extreme apex and along the anterior edge. On section, the general appearance was that of red hepatisation, the central portions being of a lighter colour than the rest, and inclining to grey. Several dark wedge-shaped patches were also seen (recent hæmorrhagic infarcts) in the neighbourhood of which the vessels were found thrombosed.

Right middle lobe: soft and crepitant.

Right lower lobe: quite solid, in appearance resembling the upper, and containing also some infarcts.

Left upper lobe: rather dark in colour, showing consolidation with considerable œdema and some infarcts.

Left lower lobe: fairly crepitant, except for a patch of consolidation at its upper and one at its lower edge.

Heart: ante- and post-mortem coagula in cavities of both sides. Muscular tissue firm.

The diagnosis was in this case at first very uncertain, and even when pulmonary signs made their appearance they were of that scattered description not incompatible with their being a part of the manifestations of typhoid fever. The enlargement of the spleen, again, pointed in the same direction; and, although there were, with one doubtful exception, no spots present, one or two relaxed pale motions were passed. At the date at which the case was observed neither the leucocytosis nor the agglutination test was known, but the extent of the pulmonary lesions led to the diagnosis of septic pneumonia, which proved correct.

Migratory, Creeping, or Wandering Pneumonia.—This variety, which has been well described by the late Professor Dreschfeld,[19] probably does not differ essentially from the septic variety, of which the case just related is an example. Here again we have a more insidious onset, a more fluctuating type of temperature, and an incomplete crisis or termination (when not fatal) by lysis. The physical signs show a consolidation involving successive contiguous portions of one lung, and then extending to the other in the same manner, or in other cases attacking different portions of the lungs. Bronchitis as a marked feature is absent. The illness is of an asthenic type, less sporadic, and more liable to attack families and groups of people under certain unhygienic conditions. Dr. Dreschfeld found in his fatal cases that sections of portions of the lung more recently attacked teemed with encapsuled diplococci, some situated in the exudation within the alveoli, others occupying the interstitial tissues. Although more numerous than in ordinary pneumonia, they were not otherwise distinctive.

Latent Pneumonia.—Cases are sometimes met with in hospital and private practice, but mostly in the former, in which, with characteristic rise of temperature and symptoms suggestive of pneumonia, often including rusty sputum, there are yet from first to last no physical signs which would warrant a diagnosis of this disease. In such cases there is probably some deep central patch of consolidation. In other instances

the symptoms are but slightly marked, so far as they point to chest disease, and only at the autopsy is the pneumonia discovered. Cases of this type not uncommonly arise at the close of malignant or surgical maladies.

Influenzal Pneumonia.—This variety of the malady, with which, owing to the recent epidemics, we are now unhappily so familiar, is essentially of the nature of a broncho-pneumonia, and will be considered in the succeeding chapter (see p. 331).

Unusual Terminations of Pneumonia.—Although as a rule the consolidation of pneumonia clears up with fair uniformity in the inverse order of its formation, it certainly does not do so in all cases, and detached islets of resolving exudation sometimes give rise to physical signs—largish clicks and circumscribed blowing sounds—which it may be almost impossible to distinguish from those of pulmonary disintegration. A careful examination of the sputa for elastic tissue and tubercle bacilli is then essential.

In other patients resolution may proceed very slowly, and the lung remains for some weeks, or even months, in a condition of consolidation. Such *delayed resolution* is most apt to occur in alcoholic subjects, and may eventually clear up. In many cases, however, the lung is left somewhat thickened and œdematous, giving rise to those permanent crepitations which are heard over the base of one lung in certain elderly persons. In exceptional instances fibrosis of the lung tissue, leading later to bronchiectasis, takes place.

Another termination of the pulmonary disease sometimes met with in pneumonia is *diffuse suppuration*, or *purulent infiltration*, of the lung (see p. 295). The symptoms which attend this fatal change are of a typhoid type: the fever continues and shows a fluctuating range; the tongue becomes dry, brown and tremulous; the prostration is marked and attended with muttering delirium; rigors, sweating, and sudamina may be present. The redux crepitations normal to the period of disease are replaced by coarse liquid râles; the respirations become increasingly rapid, and attended with laryngeal rattle, and the pulse rapid and compressible. Death soon terminates the scene.

Abscess, or circumscribed suppuration of the lung is occasionally met with. The purulent collection is usually,

although not necessarily, a small one; the walls are generally soft, and the surrounding lung tissue in a condition of disintegration. In other cases several small foci of suppuration are present. The condition, like that of purulent infiltration, is generally due to secondary invasion of the lung by the pyogenic cocci, but may result from the unaided action of the pneumococcus.

The symptoms attending the formation of an abscess are sometimes slight, but continued pyrexia and rigors, followed by sweatings, towards the latter period of the disease would suggest suppuration. But it not infrequently happens that the first sign which enables us to recognise this complication is the discharge of the abcess through the bronchus, and the sudden expectoration of a large quantity of pus, which may be very fœtid and amongst which irregular fragments of lung tissue and elastic fibres are to be found. This having occurred, cavernous breath-sound and gurgling râles will be heard over a portion of the consolidation where the breath-sounds were perhaps before suppressed. Recovery from this complication may take place, but the outlook is always serious. For a further consideration of the subject we must refer the reader to Chapter XXII.

Gangrene of the Lung is also a condition which from time to time presents itself in the later stages of pneumonia in cachectic subjects, and particularly in the intemperate. It is more frequently met with in apex pneumonia in the adult. The symptom which characterises the onset of gangrene is fœtor of breath, and there may be no other sign of the complication, for the sphacelus need not be large, and until it has been evacuated no appreciable cavity may exist. The disintegration of the sphacelus is attended with darkened fœtid sputa, which on microscopical examination will sometimes be found to contain fragments of lung tissue and elastic fibres, not, however, so frequently as might be anticipated, for reasons which we have already given (p. 73). Gangrene is a very grave complication, though not necessarily fatal.

Implication of Other Organs.—In the majority of cases complications affecting other organs are produced, as we have seen, by the dissemination of the pneumococcus, which may reach its destination either by the lymph tracts, as in empyema, or through the blood, as in meningitis, endocarditis, or arthritis,

In rare instances they are produced by the ordinary pyogenic organisms.

If we examine the statistics from the various London hospitals, brought forward in the discussion upon "Pneumonia and its Complications" at the Royal Society of Medicine, introduced by Dr. Hector MacKenzie,[20] we find that such complications are in reality uncommon. The incidence of the more important of them is shown in the following table, which we have compiled from the London statistics above referred to, and which relate to 7,394 cases of the disease, of which 1,592 cases died, giving a total death-rate of 21·51.

TABLE SHOWING THE FREQUENCY AND MORTALITY OF THE MORE IMPORTANT COMPLICATIONS OF PNEUMONIA, BASED UPON THE RECENT EXPERIENCE OF THE CHIEF LONDON HOSPITALS.

Complications.	Frequency of Occurrence among 7,394 Cases.		Mortality among the 7,394 Cases.		
	Actual.	Per Cent.	Actual.	Per Cent. of those affected.	Total Percentage.
Empyema	258	3·48	83	32·17	1·12
Pleurisy, with serous effusion	114	1·54	13	11·40	0·17
Pericarditis	243	3·28	172	70·78	2·32
Endocarditis ...	62	0·83	54	87·09	0·73
Meningitis	23	0·31	22	95·65	0·29
Peritonitis	23	0·31	16	69·56	0·21
Gangrene of lung ...	31	0·41	26	83·87	0·35
Abscess of lung ...	24	0·32	19	79·16	0·25
Arthritis	29	0·39	5	17·24	0·06
Otitis media	25	0·33	4	16·00	0·05
Venous thrombosis ...	28	0·37	4	14·28	0·05
Colitis	14	0·18	7	50·00	0·09
Parotitis	4	0·05	1	25·00	0·01
Peripheral neuritis ...	4	0·05	1	25·00	0·01

Of the above complications *pleurisy* is a more or less essential concomitant of pneumonia. When pronounced, however, it forms a serious complication of the disease, the suffering which it entails adding greatly to the exhaustion of the patient. Moreover, in cases in which the pleura is so decidedly involved the temperature does not as a rule subside with the abruptness typical of pneumonia. In some cases an effusion forms, either of the sero-fibrinous or purulent variety. In the case of a serous effusion, which is less common, the liquid is as a rule soon reabsorbed. The purulent variety (empyema) is generally heralded by a secondary rise of

temperature after the critical fall: the pus must be evacuated without delay, and if this be done, the prognosis in the majority of cases is favourable. Of the 258 cases in the above table, recovery took place in 175, or 68 per cent.

Almost as often as empyema do we meet with *pericarditis* as a complication of pneumonia, and statistics show that it is a very serious one. The danger must not, however, be exaggerated, for the inflammation may be limited to a fibrinous patch, which is discovered unexpectedly by hearing the friction sound during the daily examination of the patient, and under suitable treatment the condition sometimes clears up quickly. If extensive, the complication is an index of a grave variety of pneumonia, and the case often terminates fatally.

A purulent effusion, or pyopericardium, may develope. This dangerous complication, much more rare than the corresponding affection of the pleura, generally occurs somewhat late in the disease, and is not necessarily preceded by any friction sound, or accompanied by fever. Thus we have known it to arise after pneumonia in an abstemious and previously healthy man of twenty-four, and to be indicated only by a gradually enlarging area of cardiac dulness and an increasing irregularity and feebleness of the pulse. The temperature, which in this case had fallen to normal on the fourteenth day, did not, during the development of the cardiac symptoms, rise above 99° up to the time of the patient's death, nineteen days later. Indeed, so slight were the signs and so gradual the increase in cardiac dulness, that the case was regarded during life as one of dilatation of the heart, and it was not until after death, when the pericardium was found to be full of pus, that the true nature of the lesion was revealed.

Endocarditis of the malignant type is a rarer but more dangerous complication than pericarditis. In some cases it manifests itself by a return of fever some little time after the temperature has become normal, by sweatings, rigors, and by the development of cardiac murmurs and symptoms; but more often the objective signs are obscure. It attacks chiefly the left side of the heart, and the aortic valves more frequently than the mitral, and is more apt to occur when an old endocardial lesion is present.

Meningitis may occur either during the course of the pneu-

monia itself or very shortly after the crisis, when a secondary rise of temperature, with meningeal symptoms, indicates its presence. It is a rare complication, nearly always produced by the pneumococcus, and is speedily fatal.

Among other complications sometimes met with, may be mentioned *suppurative peritonitis*, which is always of grave augury. Of more favourable outlook are *arthritis*, which affects especially the knee and shoulder, and *otitis media*. Both are purulent in nature, and sometimes, but not always, the result of pneumococcic infection. *Suppurative parotitis*, on the other hand, is more often produced by the staphylococcus aureus.

Venous thrombosis, colitis, and peripheral neuritis are complications also met with from time to time.

Prognosis.—Each year more than 20,000 persons die in England and Wales from pneumonia, a mortality sufficiently great to place the disease in the front rank of those dangerous to life. Advanced age and alcoholism are the most important states unfavourable to recovery. Pre-existing chronic diseases, such as Bright's disease or diabetes, are likewise very unfavourable elements in prognosis. It is also a relatively fatal malady in those whose nervous systems have been exhausted by previous mental anxiety or overstrain.

The severity of attacks varies much in individual cases, and in the pneumonias prevalent in different years. At St. Bartholomew's Hospital, in the ten years 1906 to 1915, there were admitted 2,113 cases, with 255 deaths, a death-rate of 12 per cent., the mortality varying in the different years from 8·6 to 14·9 per cent. But above this percentage of fatal cases there is a highly fluctuating margin in which elevation or depression of the death-rate is greatly dependent upon treatment. In each case the extent and character of the disease must be taken into account. It will be obvious that double pneumonia is much more serious than single. The prognosis is also more grave in the septic forms which we have described, but such cases differ greatly among themselves, and we have no statistics by which their mortality can be exactly compared with that of ordinary pneumonia. The danger in these cases arises more from the general state than from the extent of lung involved. In children pneumonia generally runs a favourable course, and the consolidation melts away some-

times with astonishing rapidity. Apex pneumonia is a somewhat more dangerous variety than the basic form in that it is more prevalent amongst alcoholic and cachectic subjects.

Herpes, especially on the lips, is a not uncommon accompaniment of pneumonia. Geissler[21] observed it in 182, or 43·2 per cent., of his 421 cases, and showed that the mortality was considerably less among those in whom the complication occurred. This observation agrees with our own experience.

A temperature above 105°, a pulse over 120, and respirations over 40, are significant of a serious case. Delirium is always a grave symptom, and in intemperate subjects delirium tremens is apt to occur, especially, perhaps, in the apical variety of the disease.

A brick-dust rustiness of sputa is the usual degree of sanguineous tinge in the first days of the disease. A darker and more prune-juice colour is, however, sometimes observed in old people, and in them it is not necessarily of unfavourable augury. Decided hæmoptysis is rare in pneumonia, and was regarded by Walshe as significant of tubercle. We have, however, seen several examples in which rather sharp hæmopytsis has occurred, and in which the disease nevertheless ran a favourable course, without any evidence of tuberculous infection. The absence of any colouration of sputa in otherwise well-marked pneumonia would lead us from experience to fear a tardy or incomplete convalescence.

Treatment.—Before speaking of treatment, it is important again to observe that pneumonia varies greatly with regard to severity and type in different epidemics, and that the subjects are individuals varying, perhaps even more widely, in constitution and powers of resistance. Hence, while there may be a general plan of treatment best adapted to steer the patient through the dangers of this disease, there is room for much diversity in detail, and no one system of treatment can be accepted for all cases. Before all things it must be remembered that the physician has to deal with a general disease, a specific infection, as well as with a local expression of that disease, the inflammatory condition in the lungs.

The difficulties and dangers which arise are grouped about the four consecutive periods of the malady—those of *hyperæmia*, *consolidation*, *crisis*, and *resolution*.

Absolute confinement to bed in a good well-ventilated room

of moderate temperature, and the attendance of a careful, obedient nurse, are of the first importance in the treatment of all cases of pneumonia.

1. *Stage of Hyperæmia.*—In the first stage of the disease (*a*) shock, (*b*) pyrexia, (*c*) pulmonary congestion, and (*d*) pain are the indications which direct our treatment.

The *shock* at the commencement of the disease, immediately after the rigor, is often considerable, especially in old people and young children, although it is rarely so marked as in certain cases of acute bronchitis. Alcoholic stimulants may be needed at this period, but quietude in bed and nutritious soup are the best restoratives. A few doses of bromide with aromatic ammonia may be required.

In this pyrexial stage of pneumonia the bowels should be well cleared by a few grains of calomel and a saline aperient, after which a mixture containing liquor ammonii acetatis and citrate of potash should be prescribed. In this way blood-pressure is relieved, and pulmonary congestion lessened by natural elimination from the skin, kidneys and bowels, instead of by emptying the arterial into the venous system, as is effected by drugs of the aconite class when given in sufficient doses. These latter remedies were fashionable twenty years ago, but as the favourable issue of the disease commonly turns upon the maintenance of heart power and vessel tonicity, and since both are lowered by aconite and its allies at the very outset—sometimes, indeed, beyond recall—their administration cannot be recommended.

The *pyrexia* of pneumonia is of brief duration, and, it must never be forgotten, is normal to the disease. If, therefore, the temperature, which should in all cases be carefully watched and recorded, does not mount above 104°, it requires no energetic interference at this stage. Sponging with tepid water, to which a little sal-volatile or Rimmel's toilet vinegar has been added, will slightly moderate it, and be comforting to the patient. Cool drinks and saline medicines will be similarly useful. In children, who can be lifted about with ease, the temperature may be kept under by the warm bath at 90° employed as often as may be necessary, with small doses of quinine. In adults this measure is in private practice more difficult and disturbing to carry out. It may be replaced by "cradling"—that is to say, lifting the sheet from off the

patient on to a cradle placed over the bed, and, if necessary, suspending from its central rib india-rubber bags containing ice ("ice-cradling"). If the temperature reach 105°, a powder or cachet containing 2½ grains of quinine and 5 or 7 grains of aspirin, four or six times in the twenty-four hours, should be given to the adult. In some cases, where the pyrexia is high, and bearing in mind its real origin in bacterial toxæmia, quinine may be given per rectum in 5 or 10 grain doses in the form of suppository made with coca-butter, morning and evening, supplementary to or in replacement of that taken in the mixture.

Should the temperature rapidly rise in spite of the remedial measures above suggested, the cold pack or the cool bath—i.e., at 80° or 70°—must be employed. Ice-bags to the head are useful for adults in aid of antipyretic measures.

The *pain* of pneumonia, which indicates the seat of lesion and aggravates dyspnœa, should be treated by local rather than general measures. Opium or the subcutaneous use of morphia may be necessary in exceptional cases, but they are not desirable means of combating this symptom. Hot poultices frequently renewed, due care being taken to apply them with as little disturbance of the patient as possible,* are, on the contrary, valuable remedies in relieving pain by lessening vascular tension in the pleura. This they effect by dilating the superficial capillaries of the intercostal vessels concerned. Antiphlogistine is a convenient substitute for poultices, especially where skilled nursing is not available, since an application of the antiphlogistine paste, covered with a layer of warm cotton-wool, can be left applied for twelve hours without further disturbance of the patient, giving support and having a decided stimulating effect. When the pain is severe, from four to a dozen leeches may be employed with great relief, and it is sometimes well to encourage the bleeding by the immediate renewal of poultices or fomentations. The condition of the patient, and especially the evidence of any venous plethora, as indicated by lividity, and hardness of pulse, will guide us with regard to the number of leeches. In country or colonial practice cases may very possibly be met with in which a venesection to eight or twelve ounces may be useful at the first onset of pneumonia. In cases of severe pain,

* See direction under this heading in chapter on Bronchitis, p. 188.

where leeches are not desirable, a blister of three or four inches square, applied under the poultice, is often valuable. Cold applications are recommended by many physicians; we cannot say that we have been at all impressed with their utility, except in cases in which the fever is excessive or prolonged, when they are useful in relieving pain and reducing pyrexia.

The diet of the patient must consist of nutritious fluids, milk, strong beef-tea, mutton or chicken broth, with perhaps some farinaceous thickening. The milk may be diluted with effervescing water or flavoured with tea or coffee. Cream is sometimes a useful addition to the dietary. The food and stimulants should be given at intervals of about two hours, and in reasonable quantities, adapted to the exigences of the case. It has occurred to us not infrequently to see a patient overdone with food, his circulation overloaded, and the abdomen distended from the daily ingestion of many pints of fluid, at a time when the respiratory functions are almost in abeyance, the right heart already embarrassed, and elimination of unused material difficult. Four or five ounces of fluid food every two hours is usually ample for all requirements.

As a rule stimulants are not needed in the treatment of this stage of pneumonia, but in cases of asthenic or typhoid type, with high fever, delirium, tremulous tongue, and rapid compressible pulse, they must be prescribed in sufficient and properly regulated doses, the alcohol being given alternately with a mixture containing carbonate of ammonia. The habits of the patient must be borne in mind in estimating the quantity of stimulant required.

In cases of low type, in which the features of the disease remind one of idiopathic erysipelas, it may be good practice to place the patient at once on twenty- or thirty-minim doses of tincture of iron, with half-ounce doses of liquor ammonii acetatis, and this plan in alcoholic cases will sometimes answer without the aid of alcohol. To this prescription five minim doses of strychnia may often be added with advantage.

2. *Stage of Consolidation.*—The next period for treatment is that of consolidation of the lung. At this stage of the disease, from the fourth or the fifth day to the crisis, life is most usually threatened in severe cases by failure of the heart or hyperæmia of the sound lung, not truly inflammatory in

nature, but the result of heart failure and loss of vascular tone.

It is sometimes now advisable to substitute for the salines ammonia and bark, in other cases iron with acetate of ammonia. Excessive pyrexial symptoms must be met as before. In severe cases stimulants are necessary, and they may often be usefully combined with quinine. Two- to five-grain doses of quinine should be given, so that the patient takes from ten to twenty grains in the twenty-four hours, and as much brandy or wine as his symptoms demand.

If signs of failure of heart are observed, and often in anticipation of such arising, small doses of digitalis (five to ten drops of the tincture) should be added, either to the stimulant or to the mixture. With regard to the usefulness of moderate doses of drugs of the digitalis class in this period of pneumonia, whilst speaking from convincing experience of the fact, we would point out that their use is consistent with the most rational aim in the treatment of the disease. Salines, poultices, and leeches have the common object of lessening pulmonary congestion, easing arterial tension, and depleting the venous side of the circulation. Supporting foods and, when necessary, alcohol, quinine, and digitalis unite in maintaining heart power and tonicity of vessels at the period when these tend to fail.

In cases at this stage in which the area of consolidation is large, and in which hyperæmia and ensuing œdema of the sound lung are present, the patient is in danger from abolition of respiratory function, from failure of heart, and from exhausted nervous system. In such cases oxygen inhalations are indicated. These are given for two purposes—first, to sustain heart power; and, secondly, to maintain respiratory function. Oxygen acts in the first case by increasing the aëration of the blood circulating through the lung, of which freshly oxygenated blood the left ventricle receives the first supply through the coronary vessels. In this way heart nutrition is maintained, and removal of waste products from the cardiac muscle is promoted. For this primary purpose oxygen requires to be given early in the disease, on the first suggestion of heart failure, and in doses of ten minutes' inhalation every hour or two hours. Thus administered, oxygen has the power of lessening cardio-respiratory and

nervous fatigue in a manner analogous to that observed by Colonel Flack and others from its employment during and after aerial flights at great altitudes or of long duration, as well as after great and prolonged athletic efforts.[22] Under this treatment the pulse will often be found to diminish in frequency, to increase in force, and the patient will obtain snatches of refreshing sleep. If the symptoms are more urgent, the oxygen, as recommended by Dr. Willcox and Professor Collingwood,[23] may be made to pass through a wash-bottle containing absolute alcohol, a small portion of which, being carried over with the gas, is absorbed by the lung and conveyed directly to the heart. The stimulating effect of this procedure upon the pulse is sometimes very marked. For the second purpose, that of maintaining respiratory function over a crisis, oxygen is administered at a later period of the disease, and must then be given more or less continuously. Under such conditions of oxygen inhalations an opiate may in urgent cases be given to induce sleep, when otherwise its administration would be attended with grave danger.

Strychnia, administered hypodermically or otherwise, is of great value in stimulating a failing heart. Caffeine and theobromine come next to it. These remedies act partly by increasing arterial tone, and so retaining a larger proportion of blood in the arterial system, including the territories of the nerve centres, and partly by their indirect stimulating action upon the cardiac muscle and nerves. In some cases in which the pulse is peculiarly soft and running, pituitary extract has in our experience been used with advantage.

For the threatened pulmonary failure above referred to we have found a combination of twenty-grain doses of confection of turpentine with ether and ammonia very useful, strychnia, either alone or in combination with digitalis, being alternately given in the alcoholic stimulant. Should signs of dilatation of the right heart supervene, with marked cyanosis and failing pulse, venesection to ten or fifteen ounces may be practised.

At this period of the disease the patient is often unable to sleep, or is the subject of a busy delirium. Oxygen, as we have seen, may be beneficial in inducing sleep, and relief is sometimes given by an ice-bag resting lightly on the head,

or by a band of linen wrung out of ice-cold water and fastened round the forehead. Powerful hypnotics must on no account be ordered, but a mixture containing twenty grains of chloralamide and twenty grains of sodium bromide may be prescribed, or a drachm of paraldehyde, repeated, if necessary, in an hour. It must be remembered that these patients can in the zenith of their attack only obtain sleep in brief snatches, and the utmost care must be taken by the attendants to avoid the slightest cause of interruption of such restorative interludes.

It is a small detail to notice, but one by which much comfort may be given to the patient, especially at this stage, when the respiratory passages are dry, hot, and often sore—viz., to direct the nurse to anoint the aperture of the nostril with a little olive oil, which, by trickling into the passages, keeps them lubricated. Of much greater importance is a careful attention to the hygiene of the mouth, which should be frequently rinsed, and the tongue and gums cleansed with some refreshing antiseptic lotion. We have little doubt that neglect of this precaution is responsible for some relapses of the disease from reinfection.

At the stage of the disease now under consideration the symptoms and signs reach their acme, and the strength of the patient its ebb; the crisis is anxiously waited for, and is not unattended with dangers of its own.

3. *Period of Crisis.*—The rapid fall of temperature that occurs when crisis is well marked in pneumonia, and the copious sweating or other phenomena attending it, are frequently associated with considerable shock and exhaustion. Failure of heart and pulmonary œdema are the special sources of danger. These must be met by carefully supporting the patient, temporarily increasing the stimulants to any necessary amount, and by giving moderate (three-grain) doses of quinine in combination perhaps with small doses of digitalis. Sometimes strychnia is more useful than quinine at this period. Port-wine may often be substituted for brandy.

The dangerous symptoms associated with crisis pass in a few hours, although sometimes for a day or two the temperature remains subnormal. At the termination of crisis it is often wise to suspend medicinal treatment altogether for a time, regulating the diet to longer intervals; but until critical

discharges have ceased, no important alteration should be made. No active measures should be taken to arrest such discharges.

4. *Resolution Stage.*—During the early part at least of this fourth period of pneumonia (as we have artificially divided the phases of that disease) the same recuperative measures should be continued, strict rest in bed being still enjoined, the dietary being gradually improved, some solids allowed, and the stimulants (if any used) cautiously curtailed, and changed to port-wine or malt-liquors. The condition of the tongue will be the guide on these points.

The secretions from the kidney and bowel must be looked to, for it must be remembered that inflammatory products are being absorbed and eliminated. After a few days, simple quinine or quinine and iron tonics, or a little mineral acid twice daily, may be usefully employed. The resolution as a rule proceeds steadily and satisfactorily. In some cases, however, as already pointed out, a secondary and recurrent rise of temperature, with slight chills and sweatings, attends the process. Quinine must then be steadily continued, or, if this drug disagree, arsenic may be tried, the patient being kept strictly at rest.

After a consolidation has cleared up, it is not uncommon for there to be some return of crepitation over the seat of past pneumonia; this, which is doubtless due to local atonicity of vessels favouring a passive congestion, is best treated by the employment of tincture of iron internally. An analogous condition is not infrequently met with in acute nephritis, in the course of convalescence, which may lead to temporarily increased albuminuria without any associated rise of temperature.

Counter-irritants are occasionally useful in resolving pneumonia, and two or three grains of iodide of potassium, in combination with citrate of iron and quinine, will sometimes hasten convalescence in indolent cases.

Change of air is needed to complete convalescence, and in no disease is it more important that perfect recovery should be effected. The only conditions that need be specially observed with regard to such change are the avoidance of damp, low-lying, ill-drained localities, and the choice of places where the patient can obtain exercise on level ground. Some

patients will recover best at the seaside; others, of a more nervous temperament, often do better inland.

Sphacelus of the Lung.—In rare instances it happens that the expectoration in pneumonia becomes fœtid or distinctly gangrenous. In slight cases such antiseptics as formalin solution or eucalyptus may be inhaled, but there is no more potent disinfectant than strongly pushed oxygen inhalations. When the sloughing of the lung is extensive, and a gangrenous cavity is formed, it will be necessary to call in surgical assistance (see p. 365).

In *purulent infiltration* of the lung we cannot do more than try to support the patient by bark and ammonia, wine and food.

In *circumscribed abscess* we must endeavour to keep the abscess cavity as empty and disinfected as possible, by change of posture, and the use of disinfectant inhalations as for sphacelus, and at the same time to maintain the patient's strength. Surgical interference may again in these cases be necessary, but it should not be too hastily adopted, the abscess frequently contracting under ordinary treatment (see Chapter XXII.

In the treatment of septic pneumonia, quinine, sometimes iron, and a liberal allowance of stimulants, are our most reliable remedies.

Specific Treatment.—With the discovery of the diphtheritic serum and the recognition of its valuable curative properties it seemed reasonable to hope that a similar serum might prove of equal value in cases of pneumonia. Various *antipneumococcic sera* have accordingly been prepared, but though an encouraging case has been met with from time to time, the results obtained by their employment have on the whole been disappointing.

The recent work of the Rockefeller Institute, to which we have alluded, and the success obtained by Dr. Mervyn Gordon in the treatment of cerebro-spinal fever have, however, opened a new and more hopeful vista. Dr. Gordon[24] showed that the meningococcus, like the pneumococcus, could be differentiated by the agglutination test into four main groups or types, and that if in a given patient the type of meningococcus present was determined and the patient treated with the serum appropriate to this type, so-called "monotypical serum," then the results were highly satisfactory, the mortality being reduced

from 60-70 to 12 per cent. As in the case, however, of the monotypical antipneumococcic serum to be mentioned later, the results have been better in Type I. cases than in those infected by other varieties of the meningococcus. Apparently the presence in the serum of sufficient anti-endotoxic capacity is an essential factor.

Work on similar lines has been carried out at the Rockefeller Institute in the case of the pneumococcus with promising though less striking results. As we have seen (see p. 291), at least four types of pneumococcus have been differentiated in New York, and sera corresponding to Types I., II., and III. have been prepared. Of these, the sera produced by the injection into horses of Types II. and III. appear to be ineffective, but in regard to the serum of Type I. it is different. If patients suffering from pneumonia due to a pneumococcus of this strain are treated with the corresponding serum the mortality is much diminished. Of 107 cases thus treated eight only died, a mortality of 7·5 per cent. as contrasted with a death-rate of 25 per cent. before the serum treatment was introduced.[10f]

To be effective, the serum must be given intravenously and in large doses, so that a sufficient concentration of antibodies may be effected. An injection of 90 to 100 c.c. of serum, slowly administered, is recommended as soon as possible, this dose to be repeated every eight hours until a favourable result has been effected. On an average 250 c.c. of the serum are thus given.[10g] With these large doses symptoms of *anaphylaxis*[10h] may manifest themselves, if the patient be highly sensitive to horse serum, and in every case before injecting the serum the sensibility of the patient should be tested by means of the intradermal skin test, and even if negative, a subcutaneous injection of ½ to 1 c.c. of the serum should be given, which is effective in producing desensitisation in patients who are but slightly hypersensitive. If the skin-test be positive, showing the patient to be highly sensitive, then the serum must be given every half hour, at first subcutaneously and then intravenously, in extremely small doses, commencing with 0·025 c.c., doubling the dose at each injection. Such a thorough desensitisation is a lengthy process, but it has only been found necessary in about 2 per cent. of the cases.

Mild symptoms of *serum sickness* also manifest themselves

in nearly half the patients treated with the serum, some one to two weeks after the injections, and in about 10 per cent. the attacks are severe.

It is possible that as more potent sera are produced, obviating the necessity for such large doses, these troublesome complications may become less frequent. *It is obvious also that at the present phase of experience observations such as these could be carried out only under strict control and in hospital practice.* We have said enough, however, to show that, based as it now is on scientific principles, the outlook in regard to the serum treatment of pneumonia is more favourable than it was a few years ago, and that further developments may confidently be expected.

Following the lines advocated by Sir Almroth Wright, *pneumococcic vaccines* have also been employed. The usual procedure has been to give in the adult an initial subcutaneous injection of from 20 to 30 millions of stock pneumococcic vaccine, and to follow this up by the use of an autogenous vaccine from the patient's sputum as soon as this can be prepared. We have employed such vaccines in many cases of pneumonia, but are not satisfied that we have seen any marked improvement follow their use. If cautiously given they appear to be without harmful effect. We have also tried *sensitised vaccines*—that is to say, vaccines (by preference autogenous) previously treated by being placed for some time in contact with antipneumococci serum, after the method of Besredka, and perhaps with more encouraging results.[25]

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CHAPTER XVIII

BRONCHO-PNEUMONIA

BRONCHO-PNEUMONIA, or lobular inflammation of the lung, affects certain lobules of the organ and their associated bronchioles. The inflammatory lesion is not, however, always strictly confined to separate lobules or groups of lobules, for in some cases more diffused areas of lung become affected, until, perhaps, a whole lobe may be involved.

Ætiology.—Broncho-pneumonia may occur as a *primary disease*, apart from any pre-existing bronchitis or other antecedent condition, and as such is most frequently met with in infancy and early childhood. This variety, to which the late Dr. Samuel West[1] drew attention, is less common than the secondary form, and in its clinical features resembles closely the lobar pneumonia of the adult, and like the latter would appear to be produced by the pneumococcus.

Secondary broncho-pneumonia, whether consequent upon simple bronchitis, measles, whooping-cough or diphtheria, is also most commonly observed in infancy and early childhood, and occurs especially among the badly-nourished children of the poor. As a disease secondary to influenza, phthisis, tracheotomy, pyæmia and allied conditions, it may occur at any age.

In the great majority of cases the malady is produced by the inhalation of septic material of varied nature into the finest bronchi and alveoli. In those cases in which bronchitis is already present, the inflammation of the alveoli may be attributed to extension, or to the aspiration of infected secretions from the bronchi backwards, or their projection into the alveoli during deep respiratory or tussive efforts.

In phthisis the aspiration of blood or sputum in the course of expectoration frequently sets up in a similar manner new centres of lobular inflammation and infection. After trache-

otomy also, when the mechanism of cough and expectoration is impaired through defect in the glottis, altered blood, purulent secretions and septic matters are easily inhaled into the lungs, and give rise to lobular pneumonia.

Closely connected with the preceding is the so-called "deglutition pneumonia." In this variety, either during coma or from paralysis of the larynx, food and secretions from the mouth, or even the stomach, pass the glottis, and, failing to excite the usual expiratory efforts, reach the finest bronchi, and thus produce broncho-pneumonia. The pneumonia which sometimes follows surgical operations has no doubt, in part at least, a similar explanation, since it has been shown that during anæsthesia mucus and saliva may be inhaled into the air-passages, but the nature of the anæsthetic, the skill with which it is administered, and the disturbance of respiratory movements, also play a part.[2]

Broncho-pneumonia of a septic type may follow the inhalation of the virulent gases now used in warfare. This is especially apt to occur in the case of mustard gas—dichlor-ethyl-sulphide, $(C_2H_4Cl)_2S$, the vapour leading first to a destruction and sloughing of the mucous membrane of the trachea, bronchi and bronchioles, and then to a secondary invasion of organisms from the sloughing surfaces, producing broncho-pneumonia and sometimes death. Analogous to this are the cases in civil life in which catarrhal pneumonia has followed exposure to acrid vapours, such as the pungent smoke from a burning building.

Septic organisms may reach the alveoli through the blood, as in pyæmia, septicæmia, and in certain forms of tuberculosis. In such cases the lesions affect vascular rather than bronchial areas.

Bacteriology.—With the possible exception of the rare cases which follow the inhalation of pungent vapours, broncho-pneumonia, however arising, is always produced by the action of micro-organisms. No one organism, however, is always at fault, but under different circumstances now one and now another may produce it.

From the observations of Dr. Wollstein[3] it would appear that primary broncho-pneumonia, like lobar pneumonia in the adult, is to be attributed to the action of the pneumococcus. Broncho-pneumonia secondary to bronchitis is traceable to a

variety of organisms often acting in conjunction, among which the pneumococcus and the streptococcus pyogenes figure most prominently, and less frequently the staphylococcus pyogenes aureus and albus, the micrococcus catarrhalis, the influenza bacillus, and other organisms, as shown by Dr. Eyre's[4] extended observations. With regard to the broncho-pneumonias which occur in the course of specific fevers, we have pointed out elsewhere[5] that the inflammation may be produced by the specific micro-organism of the disease (*homologous infection*) or by a mixed specific and pyococcal infection (*mixed infection*); in rarer cases it results from the action of the pyogenic cocci or other organisms only (*heterologous infection*).

The following bacterial classification of broncho-pneumonia, based upon that which we ventured to offer some years ago, at the suggestion of the late Professor Kanthack, illustrates these points:[5]

THE BACTERIOLOGY OF ACUTE BRONCHO-PNEUMONIA.

I.—PRIMARY—produced by the pneumococcus.

II.—SECONDARY:

1. *To Bronchitis*— { Pneumococcus, Streptococcus pyogenes, Staphylococcus aureus and albus, Bacillus influenzæ, Micrococcus catarrhalis, Micrococcus tetragenus, Bacillus of Friedländer, etc. } The infection is often a mixed one.

2. *Occurring in the Course of Various Specific Infections*, such as Diphtheria, Influenza, Pertussis, Measles, Scarlet Fever, Typhoid Fever, Tuberculosis, and Plague.
   (*a*) Homologous—produced by the specific organism of the disease.
   (*b*) Mixed infection—specific and pyococcal.
   (*c*) Heterologous—produced by secondary pyococcal infection.

Morbid Anatomy.—The bronchi are always more or less inflamed, and in the secondary form, with which we are chiefly concerned, are filled with thick muco-purulent secretion. As the inflammation extends, portions of collapse are found in various parts of the lung, in others patches of inflammation; but the collapsed portions themselves are often attacked with pneumonia.

The pneumonic patch feels like a hard nodule in the substance of the lung. If of small size, it will be seen on section to be more or less conical in shape, with the base

towards the periphery of the lung and projecting above the surrounding surface, thus distinguishing it from a patch of collapse. If of larger size, the pleura over it is often involved. The portion of lung affected is dark red in the earliest stages, and then gradually assumes a greyish colour. The cut surface is soft, breaks more easily than the normal lung, and is finely granular. On squeezing it a reddish juice exudes, with perhaps a drop of purulent secretion from the central bronchus. Adjoining patches tend to coalesce, and thus the greater portion, and indeed the whole, of one lobe may become affected, forming the confluent or "pseudo-lobar" variety of the disease. In cases of septic origin, such as may occur after

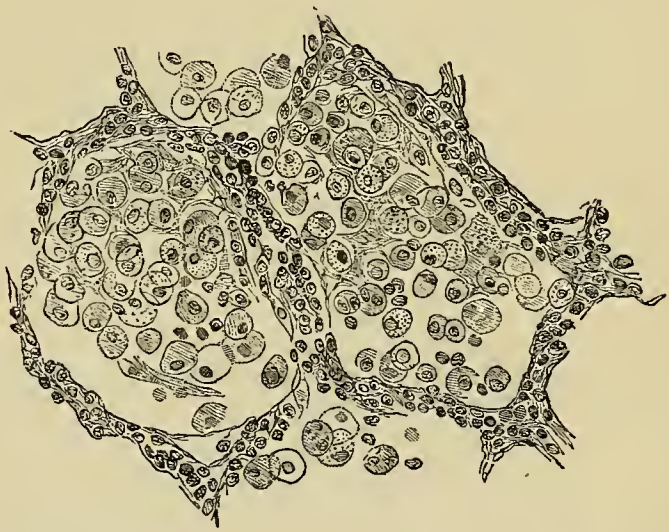

FIG. 32.—SECTION OF LUNG: BRONCHO-PNEUMONIA.

operations on the mouth and tongue, small abscesses, from central softening of the patches, are often found, surrounded by consolidated lung.

The microscopical appearances in a case of broncho-pneumonia differ somewhat, according to the acuteness of the process and the nature of the irritant. In an acute case, in its earliest stage, the appearances will closely resemble those of acute lobar pneumonia; indeed, as the late Professor Kanthack first insisted, it is anatomically incorrect to draw any hard-and-fast distinction between them. Thus many of the alveoli will be found to contain red and white blood-corpuscles, and not uncommonly a fibrin network, whilst the epithelium lining the alveoli is swollen and granular. At a later stage, or in less acute cases, with which, perhaps, we are more familiar

(Fig. 32), numerous desquamated and swollen epithelial cells, mingled with leucocytes, are found occupying the alveoli, but fibrin and red corpuscles are not visible. Later, as resolution proceeds, the cells undergo fatty degeneration, and the products are removed, partly by absorption and partly by expectoration.

Symptomatology. — Cases of *primary broncho-pneumonia* occurring in young children run a course very similar to that of lobar pneumonia. The disease sets in suddenly, perhaps with a convulsion; the temperature remains high, and falls by crisis on about the seventh day, after which the child rapidly recovers. The signs of bronchitis are less marked than in the secondary variety, and physical examination reveals small scattered areas of consolidation. The disease would appear, in fact, to be a pneumococcic inflammation of the lung analogous to the lobar disease in the adult, but which in the child takes on a lobular form.

In describing the clinical features of *secondary broncho-pneumonia,* under which are included the majority of cases seen, we shall have in our mind especially cases of infantile type, such as those arising in association with bronchitis or whooping-cough. Clinically, two forms of this disease are met with, the disseminated and the confluent.

1. *The Disseminated Form.*—In this variety the symptoms are those of capillary bronchitis, which invariably coexists. In young children the distinction between the two diseases is often impossible, and is practically of but little importance. When the disease supervenes, however, upon a less urgent bronchitis, its access is marked by a rise of temperature, an increased dyspnœa, and a greater rapidity of pulse. Shivering is rarely to be noted in young children, and its analogue, convulsion, is, so far as our experience goes, uncommon in this form of pneumonia.

The rise of temperature is generally above 102°, but although the range or average of temperature is above that of bronchitis, it is less maintained and more fluctuating in character than in croupous pneumonia. The dyspnœa, at first urgent, with flushed face and working alæ nasi, and with a characteristic "expiratory grunt," becomes less apparent as the strength of the little patient fails and his nervous centres become less sensitive in the struggle. Pallor of countenance

with perhaps a faint tinge of lividity appears, and the skin becomes moist and even perspiring. The rapidity of breathing continues, however, or increases, and on uncovering the chest and abdomen, recession of soft parts with inspiration is observed. The pulse becomes more frequent and of lessened force. The tongue is from the first thickly coated, the lips dry, the urine scanty and depositing lithates, and the bowels disordered, constipated, or, it may be, relaxed.

The physical signs of this disseminated form of the disease are not very characteristic. The percussion note is either unaltered or is somewhat raised and of semi-tympanitic quality. Auscultation reveals fine sub-crepitant or small bubbling râles scattered over both lungs, most abundant at the posterior bases. Over other parts of the lungs a patchy distribution of the râles may sometimes be recognised, and is then of value in diagnosis. The râles are persistent, not being appreciably cleared by cough, and are often better heard immediately after cough. The breath-sounds are notably enfeebled and masked. Patches of tubular breathing may sometimes be discovered.

Should recovery take place, the pyrexia, which often lasts two to three weeks or longer, gradually subsides with considerable fluctuations, the physical signs clear up, the tongue cleans, appetite returns, but strength is only slowly recovered. This form of broncho-pneumonia is, however, of a grave character, often proving fatal, and in all cases leaving behind pulmonary delicacy. Indeed, the disease may be said rarely to occur in children who are of good stock and in good previous health.

2. *The Confluent Form.*—This form of the disease, sometimes also spoken of as "pseudo-lobar," does not essentially differ in its pathology from the preceding, but the adjacent lobules of a large portion of lung, sometimes involving the whole lobe, are affected, thus producing more or less dense consolidation. This variety may be associated with ordinary bronchitis of catarrhal origin, and often occurs in the course of whooping-cough. We have also met with it in certain cases of heart disease, and as a complication in pulmonary tuberculosis.

The symptoms of the disease are identical with those of the preceding variety, except that pleuritic pain is more commonly experienced on the side attacked. The physical signs are

somewhat different. The pulse and respirations are similarly quickened, and the signs of obstructed inspiration are observed, but not symmetrically, the expansion on the affected side being, in cases in which the disease is at all extensive, notably diminished. Most commonly the posterior and lower portion of one lung is affected, and over this region the percussion note is distinctly impaired, in the earlier stages having a somewhat skodaic quality, but as the lobules coalesce becoming more toneless. Vocal fremitus in young children is of no value, and children who are seriously ill with broncho-pneumonia rarely cry; but if produced under auscultation, the cry is bronchophonic in character, with a tendency towards ægophony. The breath-sound over the consolidated area is weak and bronchial, being considerably masked by abundant sub-crepitant râles of sharply-defined or metallic character. The disease may affect both sides, but as a rule the opposite lung is affected with bronchitis only, or it may be with a few centres of disseminated pneumonia. The temperature and other phenomena in this form of the disease are the same as in the preceding.

There is a greater tendency for this variety of broncho-pneumonia to become chronic, and to run a long course of perhaps many months, terminating in pulmonary fibrosis with bronchiectasis or in pulmonary tuberculosis. The mere extent of the disease in the early stage may, however, prove fatal; in other instances it proceeds to suppurative destruction of lung and to the death of the patient. The signs of suppuration are increased prostration, adynamia, maintained rapidity of pulse, fluctuating temperature, and hectic sweatings, with rapid loss of flesh. The râles become larger, more bubbling, even gurgling, in character; and although children rarely expectorate, it is obvious that much secretion comes up to the throat, and irritative diarrhœa with slimy stools frequently supervenes as the result of it being swallowed.

Treatment.—The treatment of broncho-pneumonia is mainly that of bronchitis. In the disseminated form of the disease it is doubtful if poultices are of any service, and it is most important that the respiratory movements be allowed as free play as possible. In the more localised confluent form poulticing is sometimes useful. The timely use of emetics in whooping-cough will sometimes avert the occurrence of broncho-pneu-

monia, but it must be allowed that the disease is often started by the imprudent exposure of children with whooping-cough to cold winds under the delusion that in this disease such exposure is harmless or beneficial. A careful nurse or mother who understands how to hold a child during the paroxysms of whooping-cough, so as to permit the fullest play to the respiratory muscles, may help much in averting pulmonary collapse and subsequent pneumonia.

The room should be kept at a temperature between 62° and 65°, the air moistened, and in some cases the addition of a little tar water or the compound tincture of benzoin to the bronchitis kettle is useful. The temperature of the patient, if high, can be kept within bounds by the occasional use of the warm bath (90° to 95°), and the head may be sponged with water, cooler than this, but not cold.

The secretions must be kept clear, the patient well supported by milk, cream, and properly-made beef-tea,* and when stimulants seem requisite, they should be liberally given. In all septic cases stimulants are required. Port-wine is an excellent stimulant, and may be combined with a few minims of the tincture of bark or with small doses of quinine, or brandy may be added to the milk or broth. Careful support of the patient and good nursing are of the utmost importance in these cases.

In regard to drugs, ammonia and ipecacuanha are useful in loosening expectoration, when this indication exists. In cases, on the other hand, in which bronchial secretion is overabundant and the breathing thereby embarrassed, tincture of belladonna in two-minim doses every three or four hours is sometimes distinctly helpful. In cases associated with whooping-cough the spasmodic symptoms are also relieved by belladonna, and with such patients a few minims of the Syr. Allii Aceticus (U.S.P.) may be added to the mixture, garlic[6] being a remedy of old standing in whooping-cough.

In the convalescent stage of the disease the practitioner must bear in mind its common association with rickets and a

* Beef-tea may be made as follows: Place ½ pound of lean meat, cut up small and sprinkled with salt, in a deep dish or pudding-basin. Cover with ½ pint of cold water, and leave a couple of hours near the fire. After straining it will be ready for use. The liquid must not be boiled, or the albumin will be precipitated, and the nourishing properties of the beef-tea greatly impaired.

delicate family history, also the frequency with which remnants of disease are left behind in the lungs or bronchial glands. Cod-liver oil with steel-wine containing a few minims of syrup of the iodide of iron are now valuable, and a change of air, to a warm seaside place if possible, is very important.

Influenzal Pneumonia.

Before concluding this chapter we must make a more detailed reference to the pneumonia which is so frequent and serious a complication of influenza. In all epidemics it will be observed that a large proportion of the cases are complicated by bronchial catarrh, sometimes of the purulent type (see p. 186), or even a broncho-alveolar catarrh, of which the peculiar explosive inspiratory crepitation over one or both bases or scattered patchily over the lungs is the most characteristic sign. This may clear off, but in many instances it passes into the more grave condition of pneumonia. In influenza, as we have seen, this is of the catarrhal type, and the areas of consolidation, which in some cases are small, in others large and confluent, are set in a lung which is deeply congested, the seat of scattered hæmorrhages, relatively airless, and exuding blood-stained fluid on section. This condition of hæmorrhagic œdema is the most striking pulmonary lesion of severe influenza, and may, as Sir John Rose Bradford[7] suggests, be its essential pulmonary manifestation, following the invasion of the body by the influenza virus, possibly the filter-passing organism which he, in conjunction with Drs. Bashford and Wilson, has described. The pneumonia which follows would, on this view, be a complication due to a secondary infection by streptococci, pneumococci and Pfeiffer's bacillus of influenza, acting upon a lung already damaged by the primary disease. We must add, however, that the bacteriology of the disease still remains obscure.

In the recent epidemic of influenza Drs. Abrahams, Norman Hallows, and Herbert French,[8] estimated that pulmonary complications were present in 20 per cent. of their cases, in 8 per cent. of which the complications were of slight or medium severity, and in 12 per cent. of a serious nature, one-half to two-thirds of the latter proving fatal.

Influenzal pneumonia is always associated with an asthenic general condition; nor is this to be wondered at if we accept

the observations of Dr. Wilson[7] that the filter-passing virus is present in the blood from the early stages of the disease. In other cases a streptococcic septicæmia is present; less frequently the pneumococcus or Pfeiffer's bacillus is discoverable in the blood. This general septicæmia more frequently overwhelms the patient in the earlier stages than in ordinary pneumonia, and exercises, throughout its course, a baneful adynamic influence. The local processes, moreover, tend more frequently to pass on in severe cases to suppurative destruction of the lung. The temperature chart, as a rule, is wanting in the features characteristic of ordinary pneumonia, assuming more of the septic type. Drs. Abrahams, Norman Hallows, and Herbert French, have also noted and beautifully illustrated the peculiar lividity which they characterise as "heliotrope cyanosis," and the droop of the eyelids observed in the most grave examples of the disease, significant of the serious blood changes and the grave nervous prostration which are present. Neither dyspnœa nor undue rapidity of pulse have been marked features of the malady, but hæmoptysis, varying from a streaking of the sputum to several ounces of pure blood, has been of frequent occurrence, and is no doubt to be explained by the congested and œdematous condition of the lung to which we have referred. Epistaxis has also been common, as also some degree of nephritis, marked by albuminuria and the presence of epithelial cells and tube casts in the urine, but without œdema. The physical signs in the lungs vary with the severity of the disease and the extent of the consolidation, and are not characteristic. Such are the more important features of influenzal pneumonia, which will be sufficiently in the recollection of our readers.

The perfectly honest frankness with which Drs. Abrahams, Norman Hallows, and Herbert French confess the inadequacy of all measures to save the really desperate cases of influenzal pneumonia must not be regarded by the practitioner as a counsel of despair, and should not discourage treatment. When all is admitted, it is but to confess that influenza is a serious disease, that in grave epidemics it has a mortality of from 6 to 8 per cent., and that this mortality is principally yielded out of the 20 per cent. of cases with definite lung complications. The point in treatment is to endeavour to

prevent cases from arriving at this desperate pass, although admittedly the endeavour is sometimes fruitless.

The general lines of treatment for severe cases of pneumonia, as set forth in this and the preceding chapter, hold good, but in the light of the special gravity underlying general infection and the undoubted contagiousness of this form of pneumonia, the following supplementary measures of treatment will be found useful:

1. *Measures of Protection and Precaution against Infection.*—The patient's room should be thoroughly ventilated, properly cleansed and kept free from dust, and maintained at a temperature of 55° to 60°.

Nursing attendants should wear masks of a double fold of butter-muslin with a thin layer of alembroth wool inserted across mouth and nose.

The patient's mouth should be kept cleansed by the use of a mouth-wash several times a day, consisting of permanganate of potash, 1 in 4,000; or tincture of iodine, ʒi. to the pint; or solution of peroxide of hydrogen, 20 vol., a teaspoonful to the half tumbler; or the compound glycerine of thymol of the Pharmaceutical codex, diluted with four or five parts of water. In the case of patients too weak to rinse the mouth, the gums and tongue should be brushed over with the solution by means of a camel's-hair brush, scalded after each such use. Nurses and attendants should use similar mouth-washes and gargles as a preventive.

The sputum must be received in a vessel containing a disinfectant.

2. To combat excessive pyrexia and septicæmic incidents from 5 to 10 grains of quinine should be administered in a cocoa-butter suppository morning and evening. If the patient should be very restless, or the suppositories be rejected, ½ grain of opium may be administered with one or both the suppositories. Tepid- or hot-water sponging with eau-de-Cologne should be employed, and supplementary to this such antipyretics as aspirin 7 to 10 grains in combination with antipyrine, 2 grains, may be given from time to time as required. In some cases it is wise to prescribe considerable doses of perchloride of iron, as recommended in septic pneumonia (pp. 302, 313).

Stimulants should be freely used, a tablespoonful of brandy every three, four, or six hours as indicated.

Nourishing liquid foods should be given at intervals of two to three hours, the stimulant being mixed with the food when convenient. Tea and coffee should be allowed and cool drinks at pleasure. The primæ viæ should be cleared, and a simple lavement used from time to time to cleanse the rectum when rectal medication is being employed. Probably a neutral saline lavement is the best.

The use of oxygen is to be recommended as an auxiliary, in cases especially where the heart is disposed to fail, and sometimes in combination with subcutaneous doses of strychnia.

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[6] See "Garlic in Whooping-Cough," by T. Mark Hovell, *British Medical Journal,* 1916, vol. ii., p. 15. See also *ibid.,* p. 692.

[7] "The Filter-Passing Virus of Influenza," by John Rose Bradford, E. F. Bashford, and J. A. Wilson, with an Appendix of Clinical Notes by F. Clayton, the *Quarterly Journal of Medicine,* vol. 12, No. 47, April, 1919, p. 259.

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CHAPTER XIX

CHRONIC INTERSTITIAL PNEUMONIA OR CIRRHOSIS OF THE LUNG—PNEUMOKONIOSIS

Interstitial pneumonia is but rarely a primary disease. The connective tissue with which the lung is penetrated and clothed, which sheathes and supports its vessels and bronchi and which holds together its lobules, necessarily takes part in the parenchymatous, bronchial, and pleuritic affections of the organ; and interstitial pneumonia with but few exceptions is only met with as a consequence of bronchitis, pleurisy, or pulmonary inflammation, whether tuberculous or otherwise, and does not encroach seriously beyond the limits of the original malady. In a small number of cases it has its origin from the deposition in the lung of particles of irritating dust—a variety of the disease to which the name "pneumokoniosis" has been applied.

Chronic interstitial pneumonia is, perhaps, best seen in connection with bronchiectasis or in cases of fibroid phthisis, but is not infrequently a sequel of broncho-pneumonia, much more rarely of true lobar pneumonia. As a rule a considerable area of lung is involved, it may be the whole of one lower lobe, and sometimes also the upper lobe. In other cases, as, for example, certain of those arising from the inhalation of irritating particles, the areas of cirrhosis may be circumscribed, smaller, and scattered throughout the lungs.

The morbid changes met with in pulmonary cirrhosis are fairly uniform in all cases, and consist of condensation and collapse of the alveoli of the lung, through invasive overgrowth of its connective tissue, (*a*) spreading in branching tracts from the thickened sheaths of the bronchi or vessels towards the periphery of the lung; (*b*) extending inwards along the lobular interstices from the thickened subpleural connective tissue; or (*c*) radiating from the scar-tissue about

some centre or centres of retrogressive pulmonary lesion. In the more rare cases in which the affection follows acute lobar pneumonia resolution has failed to occur, and gradually the affected area becomes converted into a dense fibrous mass.

Microscopically it is found that enormous multiplication of the nuclei proper to the connective tissue, with a variable degree of extravasation of leucocytes, constitutes the first stage. This is followed by the conversion of the nuclear tissue into fibres (Fig. 33), at first spindle-shaped, and with nuclei still staining deeply with carmine. These nucleated fibres gradually elongate and interlace with one another to form

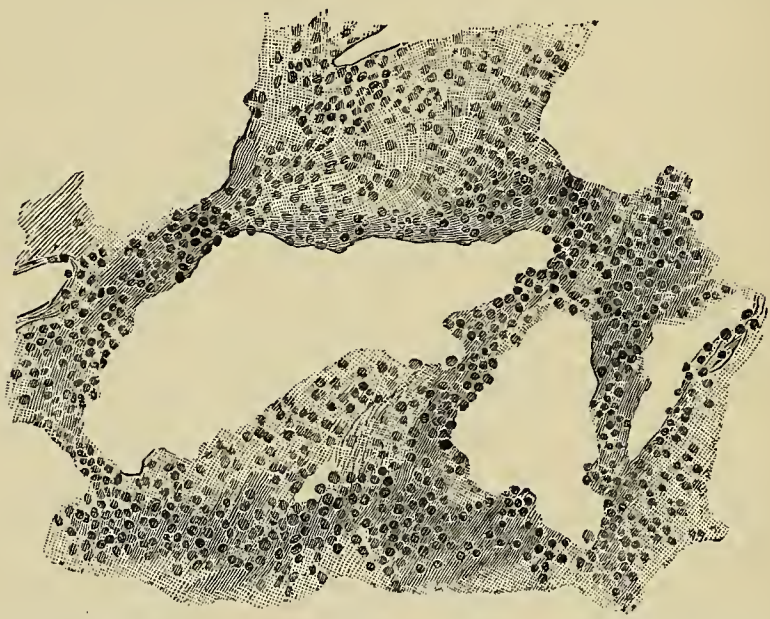


Fig. 33.—Section of Lung: Chronic Interstitial Pneumonia; Nuclear Proliferation passing into Fibrous Tissue.

fibrous tracts or bands in which the original nuclei become more scarce, and no longer take the stain well. New vessels are formed, but in no great abundance. The tissue at first, in the cellular stages, comparatively soft and pink-looking, becomes gradually more dense, opaque, pale and dry, whilst at the same time specks of pigment are deposited, giving an ash-grey hue to the whole. The various stages of connective-tissue proliferation, resulting in dense fibrous tissue, can best be observed in pleuritic thickening.

Alcoholism and syphilis, and especially alcoholism, are the conditions peculiarly conducive to the occurrence of fibroid

changes in the lung, whenever such changes are favoured by the occurrence of inflammatory or congestive attacks.

The *clinical features* of fibroid disease of the lung are necessarily somewhat varied. Condensation of lung texture, impairment of pulmonary function, and contraction phenomena are, however, essential factors in the clinical presentment of pulmonary fibrosis, no matter whether the lesion be seated at the apex or base or in the central portions of the lung, though they may be at times somewhat masked by the presence of compensatory emphysema.

Dulness and retraction, with diminished mobility of the part affected, are therefore invariable features. Flattening of the chest walls, raising of the diaphragm, and drawing of the mediastinum towards the affected side, are to be observed in varying degree in different cases. The breath-sounds are enfeebled, and their vesicular character spoiled or altogether wanting. Vocal fremitus is always diminished, and in those cases in which the extension is chiefly from the pleura it is more or less completely annulled. The contractile changes, which always ensue upon pulmonary fibrosis, produce more or less widening of the bronchial tubes, especially in those cases in which the process has extended either from the periphery, or starting from the centre has reached the periphery. In some cases these tubes are so enlarged and bulbous as to constitute bronchiectatic cavities appreciable to auscultation.

The *symptoms* are mostly of a negative kind—breathlessness, to some extent due to deprivation of lung; disturbed heart's action, from the right ventricle being burdened by the increased resistance to the circulation through the contracted lung; and cough, variable in degree according to the presence or absence of bronchitis, paroxysmal in character, and often terminating in vomiting, these features being due to the mechanical difficulty of clearing mucus from widened tubes set in rigid inelastic lung, with no air-containing lobules beyond them. Besides these general characteristics it may be said that, both as regards symptoms and physical signs, the fibroid lung gives but a modification to the clinical features of those diseases, such as chronic bronchitis, pleuritis, pneumonia, bronchiectasis, etc., of which it may form a part, and to some one of which it is generally to be ascribed.

In *diagnosis* the fibroid lung is to be differentiated from more active lesions, but, except in the case of local empyema, difficulty seldom arises, and here it can, as a rule, be cleared up by exploratory puncture. It sometimes, but rarely, happens that a bronchus becomes entangled in the contractile fibroid lesion, giving rise to signs and symptoms suggestive of aneurism or malignant disease compressing the tubes.

Pneumokoniosis.

Before leaving the subject of chronic interstitial pneumonia, that somewhat rare variety must be referred to which results from the inhalation of certain forms of irritating dust, and to which the name "pneumo- or pneumonokoniosis" has been applied.[1]

When speaking of bronchitis, we saw that it might sometimes be traced to a similar cause, and we have described (p. 264) a case of dust-bronchitis, with asthma and emphysema, which illustrated the form of disease most commonly met with amongst those engaged in dusty employments. In certain cases, however, after the bronchitis has persisted for some time, and the ciliated epithelium has thereby become damaged and its functions impaired, the dust particles find their way past the bronchi into the alveoli of the lung. Thence they pass between the cells lining the air-vesicles, and so into the lymph channels and connective-tissue spaces of the alveolar walls. Here irritation is set up, leading to the formation of fibroid material, which at first thickens the walls of the air-spaces, and later produces fibroid nodules or bands of various size (Plates XVI. and XVII.). These may again unite, until a large portion of a lobe, or indeed a whole lobe, may become consolidated.

The various kinds of dust differ much in their irritative properties, though their effects upon the lung, when ultimately produced, are pathologically of a similar nature. Coal dust is comparatively innocuous, and cases of true *anthracosis*, with fibroid changes in the lungs, are now rare, and even when met with it is probable that the fibrous changes are due to the stone dust from the strata in which the coal is embedded rather than to the coal dust itself. In these patients the expectoration is often highly charged with carbon, and is spoken of by the miners as "black spit." A simple discolora-

PLATE XVI

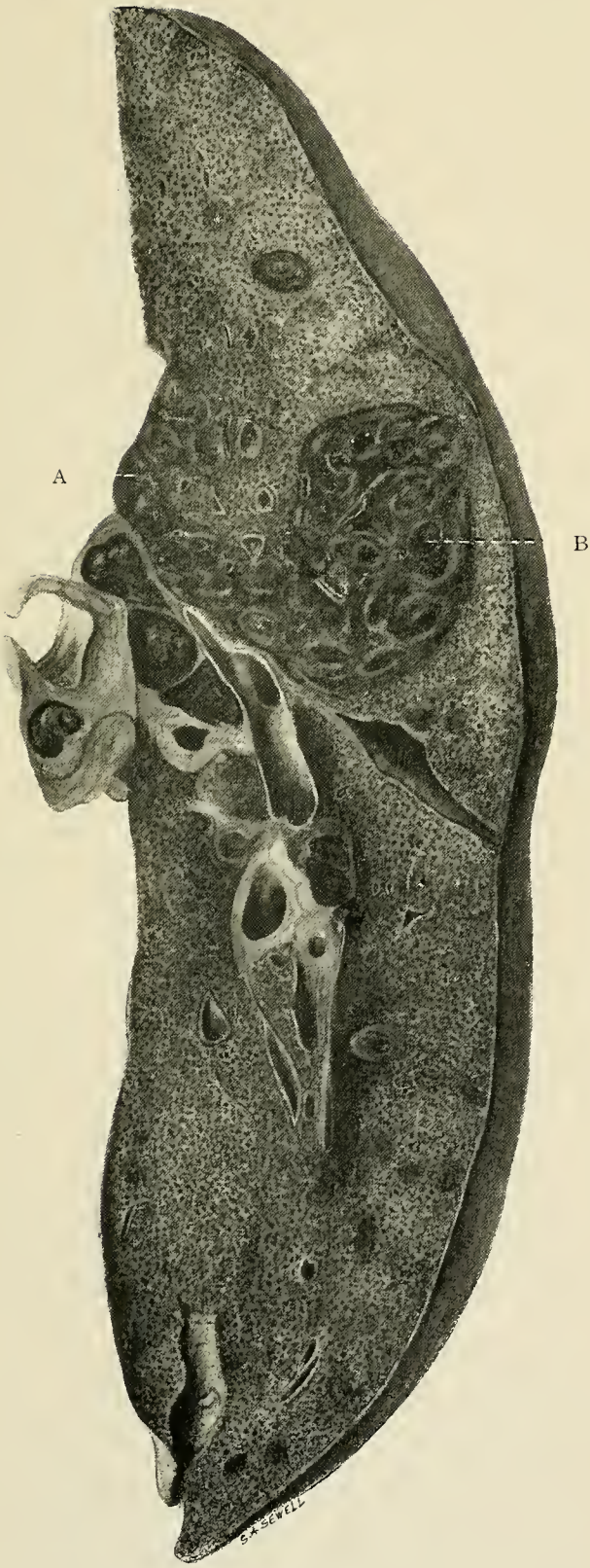
PNEUMOKONIOSIS

The drawing represents a portion of a left lung which shows the effect of the inhalation of irritating particles. The lung is denser and firmer than natural, as the result of diffuse fibrosis; and nodules of dense fibrous tissue having a bluish-black colour are seen scattered throughout it, but mostly in the upper lobe. Many of these nodules are separate and discrete, as at A; but at the lower part of the upper lobe they have coalesced into a large mass, B. No tuberculous lesions were found in the lung. The pleura is not thickened. On microscopical examination the general diffused fibrosis was found to be especially marked in the neighbourhood of the bronchi.

From a man aged forty-nine, who was engaged in the Sheffield cutlery trade, and who died of cerebral hæmorrhage.

(From the Museum of St. Bartholomew's Hospital. $\frac{2}{3}$ natural size.)

PLATE XVI



PNEUMOKONIOSIS.

To face p. 338.

tion of the lungs from deposition in them of particles of carbon is, however, extremely common in those who work in coal, as, indeed, it is to some degree in all who live in large and smoky cities. Calmette[2] and his pupils have contended that such staining is produced, not by direct inhalation, but by absorption from the alimentary canal of ingested carbonaceous

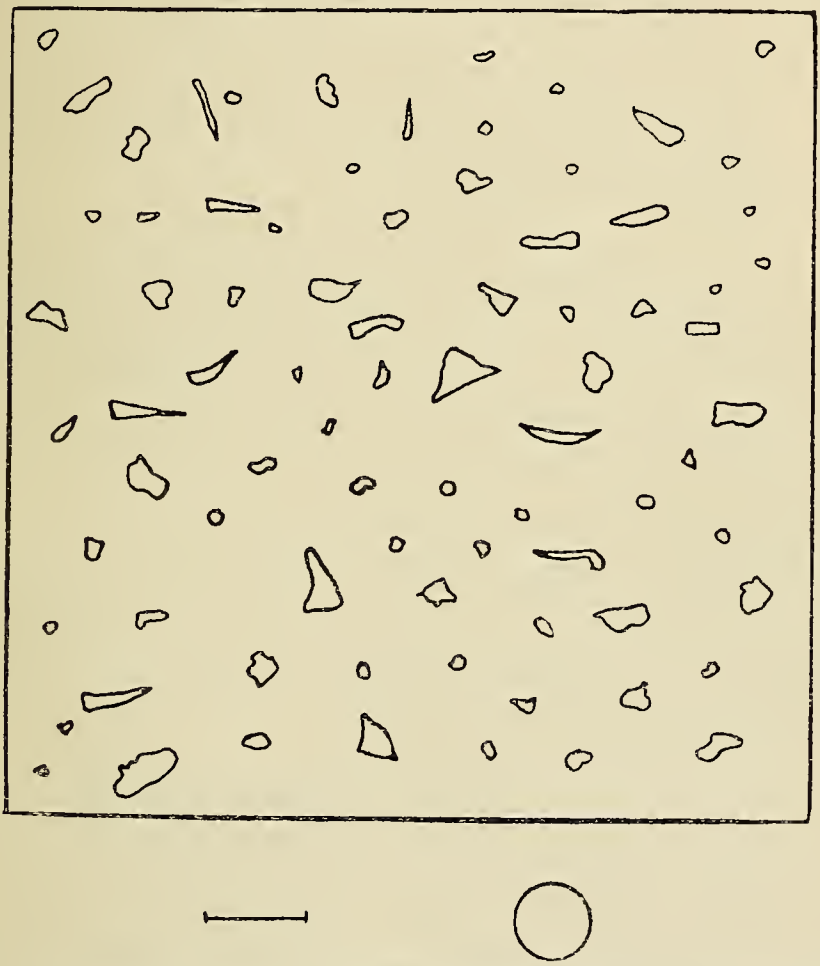


FIG. 34.—DRAWING SHOWING LARGER PARTICLES OF SILICA ISOLATED FROM A SILICOTIC LUNG. × 1,000. THE LINE BELOW REPRESENTS 10 μ, THE CIRCLE A RED BLOOD-CORPUSCLE.

(After Dr. John McCrae.[4])

particles, but his work has not been confirmed by recent observers,[3] and we may confidently adhere to the old respiratory theory as to the origin of anthracosis and, *a priori*, of other forms of pneumonokoniosis.

A much more dangerous form of dust is that consisting of fine particles of silica, which from its insolubility and the pointed and angular character of its particles, which are to be

found *in situ* in the lungs (see Fig. 34) is peculiarly irritating. To the resulting pneumokoniosis the name *silicosis* has been applied. Silica is met with geologically as quartz, quartzite, and flint, and siliceous dust is thus set free in the blasting and drilling carried on in the quartzite gold-mines of South Africa, giving rise to the disease known as "gold-miner's phthisis."[5] Silicosis is also met with among stonemasons, mill-stone builders, and potters, and among those working with ganister[6] (Plate XVII.), a hard quartzite rock used on account of its "great resistance to heat for lining Bessemer and steel converters in ironworks" (Oliver).[1a]

Particles of iron may occasionally produce similar changes in the lung tissues, but such cases of *siderosis*, as they are called, are not common. In the various forms of steel grinding it is the dust from the grindstone rather than from the steel in which the danger lies (Plate XVI.). In true siderosis the lung tissue may be of a reddish-yellow colour, from the deposition of peroxide of iron.

Such are the more important conditions under which pneumokoniosis occurs, and it is satisfactory to know that it is decidedly less frequent now than in former years, owing to the precautions which are being taken by wet-grinding, exhaust fans, and similar expedients, to protect the workman from the baleful influence of the dust.

Symptoms.—Pneumokoniosis is generally insidious in its onset. A cough developes and the breath becomes short, but there is little wasting and no night-sweating, and constitutional symptoms are not marked. The signs, in fact, are those of bronchitis and emphysema, with which the disease is generally complicated. Provided that the fibroid areas are small and scattered, as in the variety described as "generalised nodular fibrosis," there may be no impairment of note and no alteration of breathing. The emphysema may, in fact, mask the disease. An X-ray examination will, however, in many cases show clearly defined mottling (Plate XVIII.), the shadows being more sharply defined and deeper than those observed in pulmonary tuberculosis, in which the mottling has a somewhat hazy and blurred appearance.

Later, when larger areas of fibrosis have developed, the signs which we have already described at the beginning of this chapter as significant of this condition—dulness, retraction

PLATE XVII

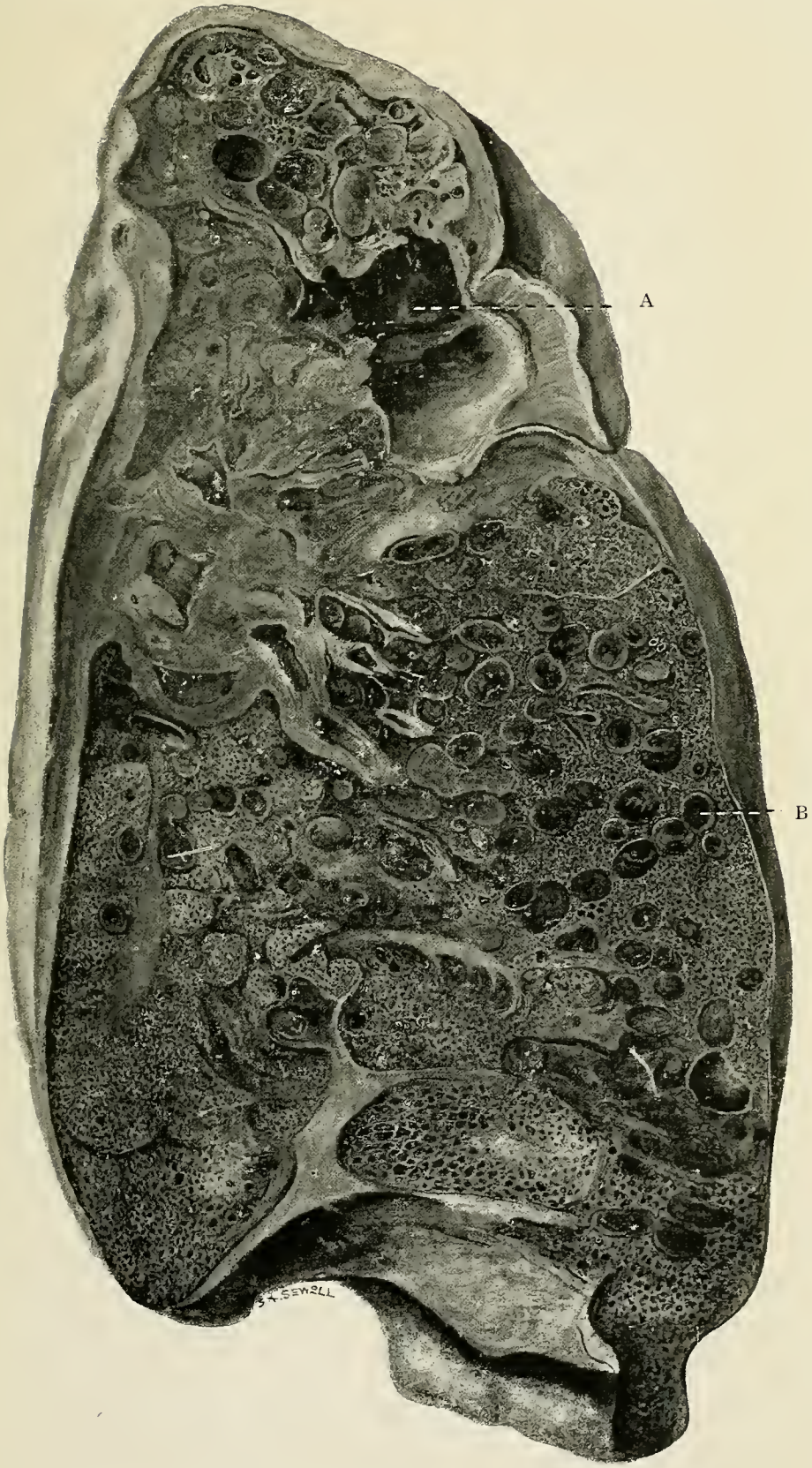
PNEUMOKONIOSIS (SILICOSIS) COMPLICATED BY TUBERCLE

The drawing shows the lung tissue to be much firmer than natural, as the result of diffuse fibrosis, which on microscopical examination was seen to commence around the bronchioles. In addition, numerous sharply-defined, hard, greyish-blue fibrous nodules, about a quarter of an inch in diameter (B), are seen scattered through the lung. Upon these lesions tubercle appeared to have become engrafted, and at A, near the base of the upper lobe, a large tuberculous cavity is found. At the apex of the lung smaller foci of tuberculous disease are visible.

From a ganister miner, who had suffered for many years from fibroid disease of the lung, and who for some time had been unable to follow his occupation. Death resulted from empyema and acute pericarditis of pneumococcal origin.

(From the Museum of St. Bartholomew's Hospital. $\frac{2}{10}$ natural size.)

PLATE XVII



PNEUMOKONIOSIS (SILICOSIS) COMPLICATED BY TUBERCLE.

To face p. 340.

with diminished mobility of the affected side, and so forth—will make their appearance. If the patient continues at his work, the disease gradually progresses, bronchial dilatation is apt to ensue, and death results, often from an intercurrent attack of acute bronchitis or pneumonia. The duration, however, varies with the nature and amount of the dust inhaled. Thus Dr. Oliver places the duration of life after commencement of symptoms at from five to six years among the rock-drillers in the South African gold-fields, and at about fifteen years among steel-grinders, while coal-miners live still longer. It has been found in South Africa that if in the early stages of the disease the miner gives up his underground occupation and takes to open-air work, arrest of the malady and great improvement in the general health not uncommonly results.

In not a few cases the disease terminates earlier, owing to infection by the tubercle bacillus, the tuberculous disease in these cases running its course with the formation of cavities, although the fibroid features still remain (Plate XVII.).

It is, perhaps, unnecessary to add that pulmonary tuberculosis of an ordinary type is frequently developed in young operatives in dusty factories, and especially among those in whom there is a family predisposition. In such the dusty atmosphere and confinement in ill-ventilated rooms strongly favour the development of the malady. These cases do not present any unusual features.

In conclusion, we may give the notes of the following case, which illustrates certain of the features of this interesting malady:

A. H. H., gold-miner in South Africa, aged thirty-two, single, was admitted into the Brompton Hospital under the care of one of us in April, 1915. He came of healthy parentage, there being no history of tuberculosis in the family, and his own health in the past had been good.

In 1902 he commenced work as a "driller" in the gold-mines near Johannesburg, his place being at the "dead end" of the mine gallery, where exposure to dust was greatest. All went well with him for 8½ years, when he began to suffer from shortness of breath and cough. These symptoms increased, and at one time kept him away from work for two months. Expectoration had never been more than slight in amount. He continued at his occupation until March, 1914, when he gave it up and came to England. Since his arrival he had lived in Kilburn, residing at home, but doing no work. His cough, however,

became worse, and a year later (April, 1915) he entered the Brompton Hospital.

On admission he was found to be a man of somewhat poor physique. Height 5 feet 9 inches, weight 10 stone 2½ pounds. He was short of breath on exertion, the respirations even at rest being generally over 20. Pulse 84; temperature normal. His cough was very troublesome, and he suffered from time to time from bouts of coughing lasting perhaps half an hour. The phlegm was scanty and viscid, and difficult to expectorate. Tubercle bacilli were never found, though the sputum was examined on four occasions at the hospital, once by the antiformin method.

On physical examination, no marked signs of emphysema were apparent, and the heart was natural. The air-entry over the front of the chest was satisfactory, but at the back the percussion note over the base of each lung was somewhat impaired, the vocal vibrations increased, the respiratory murmur weak, and numerous small râles were audible after coughing. On the left side these signs extended to the mid-scapular region. The fingers were not clubbed.

The patient was examined by Dr. Melville under the X-rays, and the characteristic findings are shown in Plate XVIII. The upper parts of the lungs are seen to be fairly free from disease, but in the lower two-thirds numerous small discrete, sharply-defined, and rounded shadows are seen, with, in places, larger and darker areas of shading, the former corresponding to scattered areas of nodular fibrosis, the latter to more extensive changes of a similar nature.

The patient stayed nearly two months in the hospital, and then returned home. He had no pyrexia, and gained 4 pounds in weight, and, on leaving, his cough was somewhat easier, and the moist sounds at the right base were fewer in number. Otherwise his condition was not materially changed.

In September he went to Eastbourne, and a month later was taken ill in a picture palace. He returned to London at the end of October, and died on November 12th, 1915, of bronchitis and heart failure, the end being preceded by distressing attacks of dyspnœa.

This case shows the long period—in this case more than eight years—which may elapse before symptoms of pneumokoniosis manifest themselves, in spite of continued exposure to the baleful influence of the dust. The characteristic X-ray appearances and the chronic nature of the malady, with fair retention of general health, are also demonstrated.

REFERENCES.

[1] In regard to the subject of pneumonokoniosis, reference should be made to :

(*a*) *Diseases of Occupation,* by Thomas Oliver, M.D., F.R.C.P., p. 298. London, 1908. Also—

PLATE XVIII

X-Ray Appearances in a Case of Gold-Miner's Phthisis of Medium Severity, showing Numerous Small Discrete and Rounded Shadows, with, in Places, Larger and Darker Areas of Shading, the Former corresponding to Scattered Areas of Nodular Fibrosis in the Lungs, the Latter to More Extensive Changes of Similar Nature.

(From the case of A. H. H., recorded in the text.)

To face p. 342.

(*b*) "The Etiology and Prevention of Pneumonokoniosis," by Thomas Oliver, M.D., being a contribution to "A Discussion on Diseases of the Lungs caused by Dust," *British Medical Journal,* 1908, vol. ii., p. 481.

(*c*) The late Dr. Greenhow's original investigations into the subject published in (*a*) *Public Health Reports* for 1860-61; (*b*) the *Transactions of the Pathological Society of London,* vols. xvi., xvii., xx., and xxi. Many of Dr. Greenhow's specimens will be found in the Museum of the Middlesex Hospital.

(*d*) "The Hygienic Aspect of the Coal-Mining Industry in the United Kingdom," Milroy Lectures, 1914, by Frank Shufflebotham, M.A., M.D., *The Lancet,* 1914, i., pp. 1731, 1799.

(*e*) "Industrial Pneumonoconioses, with Special Reference to Dust Phthisis," by Edgar L. Collis, M.B. (Oxon), Milroy Lectures, 1915, reprinted from *Public Health.*

[2] For an interesting account of Calmette's experiments, see the Cavendish Lecture on "The Etiology of Pulmonary Tuberculosis," by Sir William Whitla, M.D., LL.D., *British Medical Journal,* 1908, vol. ii., p. 61.

[3] *The Causes of Tuberculosis,* by Louis Cobbett, M.D., F.R.C.S., pp. 142-151. Cambridge, 1917.

[4] "The Ash of Silicotic Lungs," by John McCrae, Ph.D., F.I.C., South African Institute for Medical Research, Johannesburg, 1913.

[5] See (*a*) "Miner's Phthisis: Recent Investigations," by Drs. Watt, Irvine, Johnson, and Steuart, *The Medical Journal of South Africa,* vol. xi., 1916, pp. 1, 15.

(*b*) "Silicosis (Miner's Phthisis) on the Witwatersrand," by the same authors. Pretoria, 1916; and a review thereof in the *British Medical Journal,* 1916, vil. ii., p. 653.

[6] "Report on Portions of Lung from a Ganister Miner," by F. W. Andrewes, M.D., F.R.C.P., *Annual Report of the Chief Inspector of Factories and Workshops for the Year* 1900, p. 487. London, 1901.

CHAPTER XX

COLLAPSE OF THE LUNG—MASSIVE COLLAPSE

We have pointed out (p. 203) that in cases of stenosis of a bronchus, collapse of the lung to which it is tributary is apt to follow, the mechanism being that, as secretions accumulate behind the obstruction, air can no longer penetrate to the lung, whilst what remains is expelled or absorbed. In bronchitis and in broncho-pneumonia in weakly persons and in children, whose chest-walls are lax or wanting in muscular support, collapse of lobules of the lung is frequently to be observed. Mucous collection in the tubes, whilst permitting air to escape, is obstructive to its entry to the lobules, which thus gradually become deflated. In whooping-cough in young children, attended with bronchitis or broncho-pneumonia, scattered collapse of the lung having this mechanism is one of the most dreaded complications, and is one of the causes of the broncho-pneumonic centres so frequently attendant upon the disease.

Collapse of the lung from external pressure, such as from *effusion into the pleura*, of whatever kind, is more accurately spoken of as compression of the lung, although in the earlier stages the lung recedes before the effusion by virtue of its elasticity, and only later begins to be compressed. *Intrathoracic growths* may cause collapse and compression of the lung directly, or by partial occlusion of the bronchi may produce first collapse and compression, and secondly fibrosis and even disorganisation of the lung, especially in cases in which the vessels or nerves are involved. In pleuritic cases the pleura may become thickened and may bind down the collapsed lung. Still further changes may result in bronchiectasis or bronchiectatic cavitation of the lung. These conditions have been sufficiently alluded to in

connection with the diseases in which they occur, and require no further mention here.

Massive Collapse of the Lung.

There is another form of collapse of the lung which has an entirely different, perhaps more than one, origin, and which is most probably due to paretic failure of the inspiratory power of the chest-wall on one or both sides. "Massive" or lobar collapse of the lung has been defined by Dr. Pasteur,[1] who first described the condition, as a total deflation of a large area of lung-tissue of sudden onset, due to failure of inspiratory power, and attended by definite physical signs and symptoms. After referring to the papers of Professor Keith, 1903, and Dr. Halls Dally, 1908, on the mechanism of respiration, he draws the conclusion that "expansion of the lungs takes place under the influence of two forces, costal and diaphragmatic, and that the overlapping between their two spheres of action roughly corresponds to the position of the great fissure, the costal force acting mainly above and the diaphragmatic force mainly below it." Massive collapse is always attendant upon serious failure of inspiratory power, and there are two well-defined varieties—namely, (1) Cases in which collapse is due to paralysis of the essential muscles of inspiration, and is a local result of a general motor failure, as in diphtheritic or in ascending paralysis. In these cases the collapse is more or less symmetrically bilateral. (2) Cases in which the collapse supervenes suddenly without any general cause, and in which it has apparently a reflex origin, being traceable to some shock or injury to the thoracic wall, or to some more distant part. In this class the cases are for the most part unilateral. Thus the paralysis of the respiratory motor mechanism in massive collapse of the lung may be of direct or reflex (inhibitory) causation.

Group I.—This group of cases includes those first observed by Dr. Pasteur in connection with post-diphtheritic paralysis, principally in children, in which the paralysis involved the diaphragm. In these cases the lungs gradually collapse towards the fœtal position, and unless nervous power is speedily restored to the inspiratory muscles, death ensues. In such cases the deflation is more or less symmetrical, affecting

the bases of the lungs, but may involve almost the whole of one or both lungs. The physical signs, owing to the general condition of the patient, are difficult to investigate with accuracy, and the consolidation, with its signs of feeble or annulled breath-sounds and variable râles, is commonly attributed to pneumonia, broncho-pneumonia, or pleuritic effusion. Pyrexial symptoms are, as a rule, irregular; they may be wanting, and are never very marked. A notable recession of the chest-wall may be observed, whilst the arch of the diaphragm is raised, the cardiac area being likewise displaced upwards. The condition of the patient is that of general failure, and death may arise from apnœa or syncope. In some cases, however, the respiratory muscles recover their action, and rapid improvement follows the restoration of the pulmonary function.

Group II.—The interest of the cases just described lies in the fact that their observation led Dr. Pasteur to the investigation of other cases of collapse of the lung, of the second group, of greater importance, and the interpretation of which is more difficult.[1b] In these cases, which attracted much attention in connection with gunshot wounds in the recent war, and to which we have referred in Chapter X., collapse of the lung follows upon an injury, it may be to a distant part, or to some portion of the chest-wall not necessarily connected with or even on the same side as the portion of lung affected. If on the same side as the injury to the chest, the condition is spoken of as "*homolateral collapse*"; if on the opposite side, as "*contralateral.*" In other cases the collapse has followed abdominal operations. The collapse may be associated with such conditions as pleurisy, or be complicated with embolism or hæmothorax of the same or opposite side, or may pass on to some form of pneumonia; but these are complications upon which we need not in the present place dwell further.

Clinical Features.—Being commonly one-sided, the *physical signs* are more striking and conclusive than in the previous group of cases. There is flattening and diminution in size of the chest on the side affected, with depression rather than expansion during inspiration; the arch of the diaphragm is raised, and the heart displaced *towards* the affected side, unless, as in some traumatic cases, direct injury has caused an effusion of fluid on this side. The respiratory murmur,

at first weakened over the affected area, becomes more or less intensely bronchial, and is attended with crepitations and later with large mucous or even gurgling râles. On the opposite side the movements of inspiration are increased, the resonance extends beyond the normal confines towards the middle line, and the breath-sounds are exaggerated.

The collapse of the lung supervenes within twenty-four or forty-eight hours of the injury. Dyspnœa, variable in degree, sometimes very urgent, is the first symptom, and in severe cases is accompanied by more or less shock. The temperature usually rises within a few hours to 100°, or even to 102° or 103°, but is of no characteristic type. There is a short cough, rather persistent, at first dry, afterwards attended with a viscid, clear, mucoid expectoration, later becoming more abundant and muco-purulent in character. Hæmoptysis is unusual, and Dr. Pasteur states that it never occurs in pure massive collapse. Sir John Rose Bradford[2] found it a more frequent symptom in his cases, which were, however, mostly secondary to gunshot injury of the chest-wall, in the phenomena of which massive collapse is, in his experience, a frequent and an important factor. In favourable cases of reflex origin, as in those of central origin in which nerve function is restored, the symptoms gradually abate and disappear. When fatal, death is usually due to the primary nerve cause of the collapse, or to complications.

Ætiology.—It must be admitted that it is rather assumed than actually proved that paralysis of the inspiratory muscles, either as a part of some general paralysis or of reflex inhibitory origin, is the primary cause of massive collapse of the lung. The cases which originally attracted Dr. Pasteur's attention were, as we have seen, those attendant upon diphtheritic paralysis; they were mostly bilateral or symmetrical, and would seem to be of paralytic origin. The unilateral cases, occurring as they do as sequels of such lesions as surgical operations for appendicitis, hernia, salpingectomy or cholecystectomy (Pasteur)[1b]; or after such injuries as gunshot wounds, not necessarily penetrating, of the thoracic or abdominal wall, or, as recorded by Sir John Rose Bradford, of the pelvis or thigh, appear to admit no other possible ætiology than that of a reflex nervous inhibition, the precise route of which requires, however, further observation to elucidate.

Mechanism.—The pathology of the disease consists essentially of an airless collapse of the lung, which may involve the whole of one lobe or even the entire lung, or it may affect considerable portions of both lungs. Various explanations of the mechanism by which this collapse is brought about have been suggested. Of these, expiratory deflation and bronchial contraction, with absorption of air from the alveoli, are the chief.* Assuming the primary cause of massive collapse to be a paralysis of the inspiratory muscles, and more particularly of the diaphragm, the mechanism of its occurrence does not seem difficult of comprehension. The condition of the lung would rapidly be reduced to that of complete deflation, as shown in five of Dr. Pasteur's eight cases in which an autopsy was made, and in the following manner: (1) The chest-walls, being paralysed, would fall in towards the fœtal position, following the lung, which (2) would collapse to the position of elastic quiescence; (3) the respiratory efforts consequent upon the need of air would result in increased expansile movements of the unaffected side. This would have two effects—(*a*) Atmospheric pressure would bear on all sides, thoracic, diaphragmatic, and mediastinal, tending to produce more complete collapse of the affected lung; (*b*) the forcible expansion of the sound side of the thoracic cavity, whilst drawing extra air in through the main bronchus proper to that lung, would also aspirate air from the bronchus of the affected lung, and thus any residual air would be gradually but rapidly removed.

The mechanism of the second clinical feature of massive collapse—namely, displacement of the heart to the affected side (in one-sided cases unaccompanied with effusion)—is also readily explained. The same mechanism by which the chest is retracted and the lung collapsed must necessarily tend to draw the heart over to the affected side. As a clinical feature this displacement appears somewhat exaggerated, being in part apparent and relative only, inasmuch as (*a*) the thoracic cavity on the affected side is diminished and its walls approximated towards the median line: hence the margin of the chest-wall and line of the nipple are approximated to the corresponding side of the heart; (*b*) the heart is also uncovered by the

* For a full discussion of the mechanism of this form of lung collapse reference should be made to Sir John Rose Bradford's paper,[2] and to a more recent paper by Dr. J. Charlton Briscoe.[3]

Massive Collapse of the Lung with Cardiac Displacement. (After Sir J. Rose Bradford.)

To face p. 349.

MASSIVE COLLAPSE OF THE LUNG, WITH CARDIAC DISPLACEMENT

A glance at the plate, which is taken in outline from Sir J. Rose Bradford's paper[2], illustrates the approximation of the chest wall on the side affected to the median line; the uncovering of the heart on this side and its overlapping by lung on the sound side; the total effect being to exaggerate the shifting of the heart to the affected (left) side. The details are added to the original photograph.

A, heart in dark shadow.
B, lighter shadow fading to right (overlapping lung).
B′, darker than B, but less so than A.
C.C′, diaphragm.

Measurement	1—2 = 2·25	inches.	
,,	1—2′ = 2·6	,,	
,,	3—4 = 1·9	,,	opposite second cartilage.
,,	3—4′ = 2·5	,,	

The left nipple, not marked in the original plate, is hypothetically placed lower and nearer the median line in accordance with shrinking of the chest on this side.

PLATE XIX

withdrawal of the margin of the collapsed lung; (*c*) on the other side the heart's area is overlapped by the encroachment of the sound lung. A reference to the second and third radiographic illustrations of Sir John Rose Bradford's paper, and especially the third, which we reproduce (Plate XIX), shows this well.

How far bronchial contraction, of reflex origin, may be concerned in the production of some cases of massive collapse secondary to gunshot injury of the chest-wall it would be difficult to say. Colonel Elliott[3] appears to attach considerable importance to it as a cause of asphyxiative dyspnœa following upon injury. We should regard it as a separate condition from massive collapse, seeing that in asthma, the most striking clinical result of bronchial spasm, we witness not collapse, but expiratory dyspnœa and overdistension of the lung vesicles. In the local collapse of a portion of the lung, due to the occlusion of the middle division of the bronchus supplying the lower lobe, which is described by Colonel Elliott, the mechanism is separate and distinct from that of massive collapse and requires no further comment.

Dr. Charlton Briscoe[4] is inclined to attribute post-operative collapse to the half of the diaphragm concerned and its associated muscles being put out of action owing to "inflammation of the muscle or of the pleural membrane covering it," and also regards the recumbent posture as an agent in the production of the collapse. It must be observed, however, that in many cases the injury is quite distant from the affected lung, and that pleurisy is not in other cases operative in producing the phenomena.

Before concluding this chapter we may allude to another cause, which has been held to account for more or less extensive collapse of the lung—namely, the excitation of the so-called lung reflex. Dr. Abrams describes "dull areas" due to the contraction of the lung as being produced by sharp percussion of the chest surface, preferably over regions in proximity to sternum and spine. Areas of dulness thus produced are circumscribed, last but a few minutes, and are immediately replaced by resonance if similar percussion be applied over the epigastrium. This phenomenon he designated the "lung reflex of contraction." Previously he had described a dilatation of the lung which he believed to follow

"the application of any cutaneous irritant, whether mechanic, chemic, or electric," to the chest-wall.[5]

He conceives that the phenomena are due to reflex contractions or relaxations of the circular and longitudinal fibres of the musculature of the bronchi. That such contractions and relaxations of the bronchial muscles are operative in the normal respiratory function and in some of its disturbances in disease is most probable, and to this we have referred in earlier chapters (see pp. 8, 243), but we venture the opinion that these observations on the lung reflexes require more convincing repetition and demonstration. They have, however, attracted attention, and Sir James Barr[6] regards them as largely responsible for contralateral collapse, and speaks of the excitation of the lung reflexes by vigorous friction over the chest as clearing up conditions of extensive pulmonary collapse.

The *Diagnosis* of massive collapse depends, as will have been sufficiently indicated, upon the rapid development of physical signs of consolidation of the lung, enfeebled breath-sounds, contraction or immobility or depression of the affected side with, in unilateral cases, displacement of heart towards that side, and laboured and exaggerated breathing on the sound side. These signs may, however, be obscured by such complications as pleuritic effusion.

The *Prognosis* is mainly dependent upon the cause. In cases of central nervous origin, such as those following upon diphtheritic paralysis or ascending paralysis, the prognosis is grave, although by no means necessarily fatal. In traumatic cases secondary to surgical, gunshot or other injuries, the prognosis, so far as the condition of lung alone is concerned, is favourable, danger arising from associated lesions or complications.

Treatment consists in sustaining the patient, careful nursing, and the employment of remedies appropriate to the conditions with which the collapse is associated. Aeration will be usefully aided and heart power supported by the use of continuous or intermittent oxygen inhalations. Strychnia is perhaps the other remedy of greatest value.

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[1] (*a*) "The Bradshaw Lecture on Massive Collapse of the Lung," by William Pasteur, M.D. (Lond.), F.R.C.P., *The Lancet,* November 7, 1908, p. 1351.

See Also (*b*) "Active Lobar Collapse of the Lung after Abdominal Operations," by the same author, *The Lancet,* October 8, 1910, p. 1080.

[2] "Massive Collapse of the Lung as a Result of Gunshot Wounds, with Especial Reference to Wounds of the Chest," by John Rose Bradford, K.C.B., M.D., F.R.S., *Quarterly Journal of Medicine,* vol. xii., Nos. 45 and 46, October, 1918; January, 1919.

[3] Discussion of Gunshot Wounds of the Chest," by Colonel T. R. Elliott, Proceedings of the Annual Meeting of the British Medical Association, Section of Surgery, *British Medical Journal,* April 12, 1919, p. 442.

[4] "The Mechanism of Post-operative Massive Collapse of the Lungs," by J. Charlton Briscoe, M.D., F.R.C.P., *Quarterly Journal of Medicine,* vol. xiii., No. 51, April, 1920, p. 293.

[5] "The Blues (Splanchnic Neurasthenia), Causes and Cure," by Albert Abrams, M.D., Heidelberg, New York, 1908, pp. 244, 247.

[6] "The Physics of the Chest and their Relation to Disease and Injuries of the Thoracic Organs," by Sir James Barr, M.D., F.R.C.P., *British Medical Journal,* April 19, 1919, p. 471.

CHAPTER XXI

ŒDEMA OF THE LUNGS

ŒDEMA of the lungs consists of an escape of serum from the vessels into the texture of the organ and into its alveolar and bronchial spaces. As a chronic condition it is of common occurrence, and is familiar to every practitioner; but it may also occur, though much more rarely, suddenly, and in an acute form, very alarming in its intensity, and often speedily fatal.

The two forms must be discussed separately.

1. **Chronic Pulmonary Œdema.**—Of this variety there are many causes, but they range themselves naturally under two heads: (1) disturbance of circulation; (2) morbid conditions of the blood.

Thus, we may have mechanical œdema from retarded circulation, which may be due to mere feebleness of heart, or obstruction to the passage of blood through the lungs, as in emphysema, mitral stenosis, mitral regurgitation, or pressure on the pulmonary veins from tumours. Again, the powerful inspiratory efforts to draw air into the lungs through the constricted passages in croup, asthma, and other extended bronchial obstructions, cause an afflux of blood, resulting in some cases in more or less œdema of the lung textures.

In pneumonia and other acute inflammations œdema is a consequence of the active hyperæmia attendant upon the early stage of inflammation. Later, as we have elsewhere pointed out, the œdema in this complaint is not infrequently the direct result of cardiac failure, and is thus, in great part at least, mechanical in nature. It not infrequently also persists for some considerable time after the acute inflammation of the lung has passed away, owing to impairment of vessel tonicity. Pulmonary œdema may also be produced by all those morbid conditions of the blood which impair the nutrition of the vessel walls and favour the escape of serum through them.

Of these conditions albuminuria is the most important, but scurvy, purpura, and anæmia may also be mentioned.

Morbid Anatomy.—Œdematous lungs are large, heavy, wet, indented by the ribs, pitting on pressure, and on section exuding from their texture and tubes an abundant, frothy serum, sometimes blood-stained. These characters are especially marked at the bases or most dependent parts, and, indeed, may only be apparent there unless locally determined in other situations. Œdema affects both lungs, but most commonly one side is more affected than the other, owing to the posture adopted by the patient. It is very usual also to find that one or both pleuræ contain an undue amount of serum. Except at the extreme bases, and when very thoroughly waterlogged by old-standing œdema, the lungs are more or less crepitant, and portions removed will float in water.

Under the microscope, the alveoli are found filled with serous fluid, which presents after hardening a finely granular appearance, and stains faintly with eosin. Some epithelial shedding may also be observed in the alveoli; otherwise the texture of the organs is unchanged.

Symptomatology.—The symptoms of chronic œdema of the lungs are mingled with those of the other diseases of which the condition is but a consequence. Dyspnœa, straining cough, with copious thin watery mucoid sputa—sometimes, in cases of acute congestion, tinged or streaked with blood—are the chief symptoms. The patient sits up supported in bed, the respiratory movements are thoracic, "lifting" in character, the bases of the chest receding with inspiration. The front of the chest is hyper-resonant, the posterior bases more or less dull. Over the posterior aspect of the lungs, extending from the base upwards, the respiratory sounds are enfeebled or annulled, and fine bubbling râles are heard, chiefly with inspiration. These sounds closely simulate pneumonic crepitation, emphysema crackle, and the crepitation of air penetrating a collapsed lung; the distinction must be drawn by attention to associated percussion and auscultatory phenomena.

Prognosis.—Chronic pulmonary œdema is usually of grave significance. In chronic Bright's disease and in cardiac dropsy it is one of the later phenomena. In those forms of heart disease, however, which tell directly upon the circulation

through the lung a certain degree of pulmonary œdema may precede general dropsy, and may long persist without further consequences. It is very common to find a certain amount of pulmonary œdema as a permanent condition in old people with feeble hearts and emphysematous lungs, and we have met with several persons in whom slight œdema sounds have for years persisted on one side, probably as the result of a past inflammatory attack. No doubt some of these cases are explained by an œdematous condition of the connective tissue uniting the pleural surfaces over a lung that has been the seat of former inflammation. The supervention of œdema of the lungs in acute bronchitis or pneumonia is of grave augury, pointing as it does to cardiac failure.

Treatment.—The treatment of œdema from failing heart is referred to in the chapter on pneumonia. In cases of local œdema from loss of tone of vessels after inflammation, iron, mineral acids, and sometimes small doses of digitalis, are of great value.

In other cases derivative treatment is called for. The vegetable diuretics, juniper, scoparium, caffeine, and theobromine sodio-salicylate (diuretin), with digitalis, and moderate doses of the iodide and neutral salts of potash, are then valuable; and watery purgatives and diaphoretics should be used in turn. The exact nature of the case, whether renal or cardiac, will indicate the proper selection of remedies; in cardiac cases we rely more upon diuretics and digitalis, with occasional small doses of mercurial and saline aperients; in renal cases our derivative treatment is rather effected through the bowels and skin by brisk watery aperients and sudorifics, including air baths. Dry-cupping sometimes gives much relief, especially in obstructive cardiac disease, and in emphysema with dilated heart. In all cases a tonic supporting line of action is necessary.

2. **Acute Pulmonary Œdema.**—This condition, which has been recognised for many years in France, but to which until recently but little attention has been paid in this country, differs much from the chronic form which we have hitherto been discussing. In civil life it is perhaps best known as a rare sequel to paracentesis of the chest, when it gives rise to the so-called "albuminous expectoration"[1]; but, as we shall see, it occurs sometimes quite apart from pleurisy or paracentesis.

The sufferer from this malady, who may have been apparently in his usual health, is suddenly seized with extreme difficulty in breathing, and an agonising sense of impending dissolution; cyanosis follows, and very soon, in characteristic cases, a frothy exudate is coughed up, the amount being often so large and so intimately mixed with air that the foam wells from nose and mouth. On auscultation, abundant crepitations are heard throughout the chest. The duration of the attack varies. The patient may die suffocated within a few minutes, but more often the symptoms last from one to six hours, and then gradually subside. In not a few instances the attacks show a disposition to recur.[2] In other cases, after endeavouring for some time to clear his tubes, by constant efforts of cough the patient's strength fails, he becomes unconscious, and dies. After death, the lungs are commonly found in a condition of great œdema, exuding much serous fluid on section.

The expectoration is characteristic. It is large in amount, and when collected often measures one or two pints, and as much as two litres has even been recorded. After being allowed to settle, it separates into two layers, an upper frothy layer, and a lower layer, somewhat viscid, translucent, and yellowish or yellowish-green in colour, resembling in appearance the fluid removed from the pleura by paracentesis. It contains large quantities of albumin and globulin, and sometimes becomes solid on boiling,* thus contrasting with the non-albuminous sputa of pituitary catarrh (see p. 194).

If the patient recovers from the first attack—and generally he does so—he is very liable to suffer from further attacks, any one of which may prove fatal.

The malady is happily a rare one, though probably not so uncommon as is usually supposed. As Dr. Riesman[3] insists in his interesting paper, it occurs more often in those who are the subject of arterio-sclerosis, heart disease, or renal disease, and, as Dr. Leonard Williams[4] and others have pointed out, it is not infrequently associated with high blood-pressure, especially in persons of middle life or advancing years. We have seen several such cases associated with a blood-pressure

* For further details we may refer to a paper by one of us relating to this subject, in which a chemical analysis of the expectoration by Dr. Hurtley will be found recorded.[1]

of 190–225. It has been observed also in the course of typhoid, rheumatic and scarlet fever, and during influenza and pneumonia. As we have already said, it is one of the well-known, though rare, complications of paracentesis of the chest, the damaged vessels permitting increased transudation when the pressure of the effused fluid has been removed (see p. 114). In cases of extreme effusion the condition may sometimes be observed to be commencing in the other lung.

The cause of the attacks, which are apt to occur at night, and not infrequently during sleep, is not very clear. The most plausible explanation would appear to be that offered by Professor Welch,[5] of Johns Hopkins University, according to which the condition is due to a comparative enfeeblement of the left ventricle of the heart, so that it discharges, with each systole, rather less blood than it receives from the right side through the pulmonary veins. The pulmonary system thus becomes overdistended, and acutely œdematous from transudation. Such a condition, we may add, is a not uncommon contributory factor to the fatal result in angina pectoris.

Treatment.—When first called to a case of this kind an injection of morphia gr. $\frac{1}{4}$, strychnine gr. $\frac{1}{60}$, and atropine gr. $\frac{1}{100}$ should be given. The mental distress is quieted by the morphia, the heart is stimulated by the strychnine, and the pulmonary exudation perhaps to some extent checked by the atropine. In some cases, especially those in which the blood-pressure is high, venesection to ten or twelve ounces is indicated. On the cardiac theory this should give relief to the exhausted heart, and in practice it has been found to do good. It may in some cases be supplemented by leeches or dry-cupping of the chest. If the heart is beginning to fail, diffusible stimulants or strychnine subcutaneously must be freely administered. Inhalations of oxygen will also be found useful, but the oxygen must be given continuously during the serious symptoms. In cases with recurrent attacks a course of diuretics may be prescribed, on the assumption that some toxic factor may be at work, and with a view to assist elimination by the kidneys.

Irritant Gas Poisoning.

Although acute œdema of the lung, as we have seen, is uncommon in civil practice, it is otherwise in war since the

introduction of poison gas in April, 1915. Both chlorine and phosgene ($COCl_2$) are pulmonary irritants, and when inhaled they produce an acute pulmonary œdema of inflammatory nature, which is often fatal.[6]

On first exposure to the gas the soldier at once experiences a catching of the breath and choking, and a feeling of inability to breathe freely. Vomiting and cough soon follow, and after an interval, which may vary from a few minutes to some hours, corresponding to the concentration of the gas and the duration of the exposure, inflammatory reaction occurs in the lung-tissue, leading to acute œdema and the appearance of the characteristic albuminous expectoration. Cyanosis soon manifests itself, or with phosgene more often a pallid type of asphyxia, symptomatic of cardiac failure. If the patient survives for forty-eight hours, he will usually recover, though convalescence may be tardy, and some degree of bronchial catarrh is apt to persist.

Treatment must be on lines similar to that advised in cases occurring in civil practice, morphia, however, being withheld, unless urgently demanded by the patient's anxiety and distress. In view of the conditions of active service, it must be especially remembered that *rest* and *warmth* are fundamental factors in the treatment, each helping to diminish metabolic activity, and thus to lessen the need for oxygen from which the patient is suffering. Venesection, if the case be a cyanotic one and the right heart dilated, and oxygen inhalations, with strychnine and diffusible stimulants when required, will again constitute our main lines of treatment.

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[5] (1) "Zur Pathologie des Lungen Ödems," von Dr. W. H. Welch, aus New York; *Archiv für Pathologische Anatomie und Physiologie und für Klinische Medicin.* Herausgegeben von Rudolf Virchow. Berlin, 1878, Band lxxii., p. 375. See also—

(2) "Edema: a Consideration of the Physiologic and Pathologic Factors concerned in its Formation," by S. J. Meltzer, M.D., *American Medicine,* p. 195. Philadelphia, U.S.A., 1904.

[6] For a study of gas-poisoning reference should be made to

(*a*) The Reports of the Chemical Warfare Medical Committee, published by the Medical Research Committee, Nos. 1-8, 1918, and the Atlas of Gas-Poisoning (1918) which accompanies them. See also

(*b*) "Gas-Poisoning: Pathological Symptoms and Clinical Treatment," by Leonard Hill, M.D., F.R.S., *Transactions of the Medical Society of London,* vol. xxxix., 1916, p. 114.

CHAPTER XXII

ABSCESS AND GANGRENE OF THE LUNG

Abscess of the Lung.

CIRCUMSCRIBED suppuration of the pulmonary tissues may arise from a variety of causes, the most important of which are acute inflammation, whether lobar or broncho-pneumonic, especially in the subjects of diabetes or chronic alcoholism; wounds of the thorax; lodgment of foreign bodies; circumscribed gangrene; pyæmic infarctions and other infective emboli. It may also arise from extension of suppuration from adjacent parts, *e.g.*, suppurating bronchial glands, mediastinal abscesses, empyema; or it may result from a malignant growth of the œsophagus ulcerating into the lung.

In acute lobar pneumonia abscess formation is rarely met with except in broken and debilitated constitutions, and a history of long indulgence in alcohol is usually to be obtained. The abscesses are generally of small size, numerous and irregular in shape, though sometimes a single larger abscess may be found.

Broncho-pneumonia is more frequently followed by suppuration than is true lobar pneumonia. The broncho-pneumonia is generally septic in nature, produced by the passage over the insensitive glottis of acrid discharges from suppurating wounds or cancerous sores in the mouth, or of decomposing particles of food when the patient is in a state of coma. The areas of suppuration are usually multiple and small.

Multiple abscesses are, however, most commonly produced by infective emboli, especially in pyæmia. These set up areas of acute septic lobular pneumonia, which rapidly undergo suppuration, giving rise to small abscesses, which are often thickly disseminated throughout both lungs.

Symptoms.—The formation of pus in the lungs, as in other organs, is usually attended with severe rigors and pyrexia

and with symptoms of great prostration. The appearance of the patient, the high temperature, and rapid breathing, suggest that the lung is the seat of inflammation, but the first evident symptom of an abscess may be the sudden expectoration of pus with fragments of lung tissue.

Physical Signs.—If the abscess be of any size, and communicate with a bronchus, the signs observed will be dulness on percussion, with gurgling râles and the other indications of cavity. In more minute and scattered abscesses the signs are often indefinite and slight, or may be those of softening of the lung. To X-ray examination the abscess will produce a shadow, more characteristic if, as is sometimes the case in larger cavities, much air be contained as well as pus.

Treatment.—The treatment of pulmonary abscess consists generally in the pursuance of the remedies for the diseases that may have led up to it, and particularly is this the case with those grave forms of disseminated abscesses associated with pyæmia. Quinine or bark and mineral acids are useful. Disinfectant inhalations are also of service, and in circumscribed cases especially the patient should be encouraged to evacuate the contents of the abscess cavity from time to time. Under such ordinary measures of treatment, with good ventilation and change of air at the earliest moment, abscess of the lung sometimes heals, perhaps more frequently than is generally supposed.

Surgical Intervention.—In other cases the question of operative treatment by opening and draining the abscess must be considered. This may be recommended if definite signs of localised abscess cavity are present, and if after a period of observation there be no amelioration in the patient's symptoms, as indicated by a fall of temperature and a lessening of the expectoration. Much depends upon the seat of the abscess, basic cases, owing to the difficulty of natural drainage, more often requiring interference.

The records dealing with cases of this kind treated surgically by incision and drainage are most encouraging. Two successful cases were published in 1889 by Dr. S. Smith[1] and by Mr. J. D. Harris,[2] and these are among the earliest cases recorded in England. The recent figures of Garré and Quincke[3] show that out of 182 cases treated by operation, 148 were cured and 34 died, a mortality of 17·5 per cent.

(1) *To Define the Outline of the Cavity.*—The position and outline of the cavity are first defined as accurately as possible, both clinical and X-ray methods of examination being employed. For this purpose a circle should be drawn upon the chest to include the centre of greatest intensity of physical signs. Then, using a stethoscope with a small chest-piece, this centre should be gradually approached from all sides, and a mark made at each point where pectoriloquy and cavernous breath-sound are first distinctly recognisable. In this way an outer circle is drawn, marking off a larger area. Further, the limits of dulness should be defined, and the position of other organs, the heart and diaphragm especially, taken into careful account.

(2) *Exploration.*—The physician having thus marked out the area for exploration, and having controlled his results as far as possible by X-ray examination, a puncture should be made with an exploring needle-syringe, with not too fine a needle, through the central part of the inner circle, pushing the needle boldly in and quite vertically to the surface at the spot chosen. Several points may be thus explored should the first puncture not be successful.

The sign of success is the withdrawal of purulent material, showing that the cavity has been struck; and the character of the fluid, whether fœtid or otherwise, can now be ascertained. The instrument should be introduced, an inch and a half or two inches in the first place, and the syringe exhausted; if no matter appears, the needle should be pushed in farther, and then slowly and cautiously withdrawn, the operator testing each depth from the surface, and also endeavouring to ascertain by lateral movements the resistance of tissues and degree of fixity of the instrument. The length of the exploring needle should be known beforehand, so that its depth of penetration may at any time be calculated. In the event of failure to strike the cavity, it is probably best to desist from further measures, but a fearless and thorough exploration should be made at several points.

Further Steps in the Operation.—If the withdrawal of even a few drops of discoloured fluid or pus show that the cavity has been reached, the needle may be retained *in situ* as a guide or withdrawn at the discretion of the surgeon. A portion of two ribs, one above and one below the site of

puncture, should next be excised, care being taken not to wound the pleura. Since it is impossible to say whether the pleura is adherent or not, a circle of stitches two inches in diameter, as recommended by Sir Rickman J. Godlee,[4] to whose writings we must refer the reader for further surgical details, should be inserted around the site of puncture, uniting the pleural layers and penetrating the lung to the depth of half an inch. In this way the cavity of the pleura is completely shut off. The pleural layers are then divided, and if the needle has been withdrawn, the position of the pus is again localised by the exploring syringe. Sinus forceps are now passed along the directing needle until the pus is reached, then opened out and withdrawn, so as to rend open the cavity with the least risk of serious hæmorrhage. The finger can now be inserted, the cavity thoroughly explored, its dimensions in different directions ascertained, and a sufficiently large drainage-tube introduced.

Smart hæmorrhage will sometimes occur during this operation, as might be expected, but on air being freely admitted it usually soon ceases; if not, the wound must for a short time be plugged. We have not yet seen more than enough bleeding to cause momentary anxiety. Still, it is most desirable to avoid as much as possible the use of cutting instruments in dealing with the lung.

In some cases, although the exploring syringe gives evidence of having reached the cavity, it cannot be located at the operation, the needle having perhaps become displaced. Under these circumstances it is best to leave in a drainage-tube in the hope that the cavity may finally discharge through it. This happened in a successful case of Dr. Cayley's, which was operated upon by Mr. (now Sir Alfred) Pearce Gould.[5]

The treatment subsequent to the operation must be carried on in accordance with ordinary principles, and the special circumstances of each case.

Gangrene of the Lung.

Death of a portion of the substance of the lung may occur under two forms—(*a*) circumscribed; (*b*) diffuse.

(*a*) The *circumscribed* form is that usually seen, the gangrenous area being distinctly defined, and varying in size from that of a nut to a considerable patch involving the

greater part of one lobe. The lower lobes and superficial parts of the lungs are most frequently affected. The necrosed portions of the pulmonary tissue become moist, soft, pulpy, of a bluish-green or blackish colour, and evolve a peculiar and highly offensive odour. These portions consist of altered lung elements and blood-corpuscles, together with amorphous débris. The limits of the dead tissue are indicated by a zone of hyperæmia and consolidation. The necrotised lung may slough and be discharged through a bronchus, leaving a ragged cavity behind. Frequently, however, the pleura is involved, and the fœtid material may find its way into the pleural cavity, unless, as sometimes happens, adhesions have previously formed between the pulmonary and parietal layers.

(*b*) In the *diffuse* form there is no line of demarcation between the dead and healthy tissue, but inflamed, congested, and gangrenous lung are all mixed up together. The greater portion of one lobe, or of an entire lung, may be involved, or perhaps both lungs may be affected at several points.

Ætiology.—Necrosis of a portion of the lung is the result of obstruction of vessels and deprivation of blood-supply. It may be secondary to inflammation of the pulmonary tissue, however produced, or be merely mechanical in origin as from embolism of a branch of the pulmonary artery. Gangrene, with its characteristic signs, implies the presence of secondary putrefactive changes in this necrotic area.

Gangrene may sometimes arise as a complication of acute pneumonia, especially when occurring in drunkards or those suffering from diabetes. Pyæmic emboli, or the inhalation of fœtid discharges from a cancrum oris or malignant growth in the mouth, or the bursting of a neighbouring abscess into a bronchus, may also give rise to it. In the insane it is not infrequently observed, possibly from the inhalation of food during its passage over an imperfectly sensitive glottis; and for a similar reason it sometimes occurs after tracheotomy. Retention of putrid bronchial secretion, with secondary septic broncho-pneumonia, may also produce it, as seen in bronchiectasis. Gangrene may also be caused by wounds of the chest, and the lodgment in the lung of foreign bodies, such as bullets and pieces of cloth. It may complicate such debilitating diseases as smallpox, typhus fever, and the like, when the condition may be generally traced to thrombosis of a branch

of the pulmonary artery. Pressure of mediastinal growths and aneurism, which interferes with the circulation through the lungs, is another cause. Whatever the preceding conditions, it must be repeated that thrombosis or embolism blocking a branch or branches of the pulmonary artery is the penultimate occurrence.

Bacteriological examination reveals the presence in the gangrenous tissue of numerous varieties of micro-organisms. Among those that may be found we may mention the pyogenic cocci, whether staphylococci, streptococci or pneumococci, the bacillus proteus vulgaris, bacillus coli, bacillus pyocyaneus, and many others, among which may be included various anaerobic organisms. In several cases it is interesting to note that acid-fast bacilli, allied to the tubercle bacillus, have been discovered.[6] These organisms no doubt all play a part in producing the inflammatory condition which leads to the death of the lung tissue, or in originating the secondary putrefactive changes; it is evident that no one organism can be held responsible for the condition.

Symptoms.—The most characteristic symptoms of gangrene of the lung are the peculiarly offensive odour of the breath associated with the copious expectoration of fœtid and discoloured sputum containing lung tissue. Fœtid empyema, post-pharyngeal or other fœtid abscess, might possibly be confounded with gangrene of the lung; but the condition most likely to be mistaken for it is old-standing bronchiectasis suddenly becoming fœtid from necrosis of portions of mucous membrane, and from this it is to be distinguished by the previous history of the case and the presence of elastic fibres in the sputum. Such fibres are not, however, invariably found in gangrenous sputum, for reasons which we have already explained (see p. 73).

The general symptoms are extreme depression, asthenia, and collapse, and the termination is usually fatal. Hæmoptysis from the opening up of bloodvessels is not uncommon, and we have known it to be the direct cause of death. Fever is more or less of the irregular suppurative type.

Physical Signs.—In the early stages the auscultatory signs are indistinct and not to be relied upon; but later, if the gangrene be circumscribed and near the surface, the physical signs are dulness over the affected area, with gurgling râles,

accompanied by an amphoric quality of breath-sound. The distinctness of these signs varies, however, according to the position of the cavity and its degree of freedom from shreddy gangrenous tissue. X-rays, as with pulmonary abscess, may sometimes give an indication of the cavity, but here again much depends upon the presence of air as well as pus within the cavity and upon the condition of the surrounding lung, whether much consolidated or not.

Treatment.—This should be nourishing and stimulating, alcohol being freely given. Antiseptics internally are of little use; but inhalations of creosote, carbolic acid or eucalyptus, such as that suggested in an earlier chapter (p. 218), should be employed. Under such treatment in a certain proportion of cases the sphacelus may be expelled through the bronchial tubes, the cavity contract and the patient make a good recovery. But such instances are unfortunately rare, and in cases of circumscribed gangrene with symptoms of hectic, in which the locality of the gangrenous cavity can be fairly defined, surgical treatment should be attempted.

If operation be performed early, and the cavity be opened and drained in the manner already described (see p. 361), the outlook is greatly improved. Thus, in his address at the French Surgical Congress as far back as 1895, M. Reclus[7] pointed out that, whereas the mortality from gangrene, if untreated, was probably not less than 75 per cent., the figures after operation showed a death-rate of only 38·5 per cent. among 83 cases treated. The more recent figures of Garré and Quincke[3] are still more favourable; of 281 cases, 197 were cured, with 84 deaths, a mortality of 29·3 per cent.

It is to be observed, however, that the cases which would be selected for operation are not consecutive cases of gangrene of all sorts, but those, such as we have spoken of above, in which the cavity is fairly restricted and defined. Nevertheless, the figures indicate that in suitable cases surgical intervention is both justifiable and proper.

In cases of diffused gangrene surgical treatment is of no avail.

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[7] "Chirurgie du Poumon (Plèvre exceptée)." Rapport par le Dr. Paul Reclus, Neuvième Congrès Français de Chirurgie, Paris, 1895, p. 50. A translation of this article, though without the tables, will be found in Mr. Stephen Paget's work, *The Surgery of the Chest,* p. 443. Bristol and London, 1896.

CHAPTER XXIII

HYDATID DISEASE OF THE LUNGS

HYDATID disease, though rare in the United Kingdom, is prevalent in Australia, New Zealand, Iceland, the Shetland Isles, and in the Argentine. Next to the liver, which is affected in more than half the cases of hydatid disease, the lung is most frequently the seat of the parasite.* In Australia the lung is attacked in about 16 per cent. of the cases reported.[1a] In Europe the proportion is not so high, Neisser's statistics of 900 cases placing the percentage of the pulmonary variety at 7·4,[1a] and in Iceland it is said to be even less.[2a] In the British Isles the lung is rarely affected, and chiefly amongst those who have resided in Australia and New Zealand.

The disease is most prevalent in the male sex. It may occur at any age, although it is rare below the age of ten and above that of fifty. It is more frequent in the right than in the left lung; this preponderance in favour of the right side being most marked amongst cases of basic distribution. Sometimes the acephalocysts are multiple, and affect both lungs. The bases or central portions of the lungs are most commonly affected, but one or both apices may be attacked. The cysts may be found entire or broken in the bronchial tubes on their way to expulsion; they have also been found in the pulmonary arteries and right side of the heart, having been conveyed there from some distant point. The pleural cavity is rarely the primary seat of hydatid.

The source of the disease, as of hydatids in other organs, is the introduction of the ovum of *Tænia echinococcus* discharged from the alimentary canal of the dog. In England

* We are greatly indebted in our remarks on "Hydatid of the Lung" to the writings of the late Dr. Davies Thomas, of Adelaide, and also to the lectures of Dr. A. A. Lendon, of the same State.

this small parasite, the total length of which is only one-quarter of an inch, is extremely rare. In Australia, on the contrary, the late Dr. Davies Thomas found it in nearly 40 per cent. of the stray dogs examined by him,[3a] the parasite having been acquired by their feeding on the offal of sheep or cattle, the most common intermediate hosts, infected with the disease in the bladder-worm stage. The ovum usually effects its entrance into man through drinking-water, finding its way to the lungs from the digestive canal, after passing *en route* through the liver and the right heart. The above facts explain the great prevalence of hydatid disease in Australia, and especially in the country districts of the southern portion of the Continent, for there we find countless herds of cattle and flocks of sheep, as well as innumerable dogs, many of them already infected; while the water-supply, consisting often of unprotected surface pools, is not infrequently open to direct contamination by the dogs themselves. All the conditions necessary for the spread of the disease are thus at hand.

The morbid anatomy of an hydatid tumour of the lung is after the same pattern as that of similar cysts elsewhere, the external investment being furnished by the more or less smoothed, thickened, and condensed pulmonary tissue. The investment of the pulmonary hydatid is thus in the lung very vascular, and with it one or more bronchial tubes may communicate. In the lung more often than the liver the cysts are sterile and contain no daughter cysts.

Symptoms.—The symptoms and signs of hydatid of the lung may be conveniently divided into: (1) those presented by the tumour before rupture; (2) those which occur during and after rupture.

1. *Before Rupture.*—Unbroken cysts which have not attained considerable size may give rise to no recognisable signs; and so slow and insidious is their growth that even large tumours may exist for a long time unsuspected. Cough, hæmoptysis, pain, and dyspnœa, are the chief symptoms that may be present in this stage, and may lead to a mistaken diagnosis of phthisis.

The cough is dry, teasing in character, sometimes attended with slight bronchial expectoration; occasionally it is distinctly paroxysmal, and has a laryngeal croupy character. Hæmoptysis, although often met with at this stage, is usually

PLATE XX

X-Ray Photograph of a Case of Hydatid Disease of the Lung, showing the well-defined Rounded Shadow so suggestive of this condition.

To face p. 369.

but slight in amount, from a mere streak or staining of the sputum to a teaspoonful or so, and is due to active pulmonary congestion set up by the growing parasite in its immediate neighbourhood.

Pain is not felt except when the cyst has approached near enough to the surface to involve the pleura; but before this period an ill-defined uneasiness may be experienced. The dyspnœa is inappreciable except in the case of a large cyst, or one situated near the root of the lung. The centric pressure symptoms of an hydatid are, however, never very marked.

The physical signs before rupture may, in cases of small or deeply-seated cysts, be completely obscured. Over tumours of larger size, and nearer the surface, a certain degree of fulness is to be observed, with effacement of the intercostal spaces over a limited area. Percussion dulness, having a very definite and circular outline, is obtained over this region, beyond which there is normal or modified pulmonary resonance. A certain degree of elasticity may be appreciated on percussion over the most central point, amounting possibly to "hydatid thrill," a rare sign, however, in hydatid cysts of the chest, and in no way pathognomonic, since we have known it well marked at the superior level of the fluid in pyopneumothorax, and perfectly elicited in a case of hydronephrosis. Over the region of dulness vocal fremitus and respiratory murmurs are enfeebled or absent; and in some cases pleuritic friction is present over the tumour. Displacement of the heart and other organs may occur.

In association with these more or less positive signs and symptoms there are the important facts of the absence of pyrexia, the but slight interference with general health and nutrition, and the very insidious and ill-defined onset of the disease. The position of the physical signs is sometimes of value in diagnosis, when they present in the mammary or axillary, infrascapular, or some other unusual situation not commonly affected by more ordinary lesions. Of great assistance also is an X-ray examination in disclosing the hydatid, the cyst commonly appearing as a circular shadow clearly defined from the surrounding lung, and presenting a picture which is almost diagnostic (see Plate XX.).

A blood examination often shows a leucocyte count within the normal, but revealing an increased proportion of eosino-

phile cells, from 6 per cent. upwards. Such an eosinophilia is not, however, invariably present, nor is it of itself diagnostic, since it is observed in the presence of other animal parasites, as well as in asthma and certain diseases of the skin. The complement-fixation test,[4] a method of diagnosis upon the lines of the Wassermann reaction for the detection of syphilis, is as a rule positive in echinococcus disease, but even with the best technique a similar result has occasionally been observed in the absence of such affection. A negative reaction also does not exclude the presence of the disease, and should be expected if suppuration of the cyst has taken place. The reaction, therefore, cannot be entirely relied upon.

The removal through a fine trocar of the characteristic hydatid fluid of low specific gravity (1005-1007), rich in chlorides and free from albumin, containing possibly some hooklets, would solve the diagnosis. *This step, however, should never be taken until the surgeon is prepared for the rapid evacuation of the cyst*, and this for reasons to be presently mentioned.

The following cases illustrate well the main clinical features presented by hydatid of the lung before it has ruptured:

Case 1.—Mr. G., aged about thirty, came under the observation of Dr. Douglas Powell on October 14, 1891, on account of a cough and sanguineous expectoration. He was a well-developed spare man, who had lived most, if not all, of his previous life in Sydney. He had never been robust, but since he had come on business to this country in 1887 his health had improved. Two years previously he had influenza, with "catarrh of the right lung," for which he went to St. Leonards, and it was there stated to him, by the late Dr. Cooke, that his right lung was weak, but not definitely diseased, and on examination of his sputum no tubercle bacilli were found. He regained health, and remained well through the winter of 1890, although he again found it expedient to spend six weeks of that winter at St. Leonards, his own home being in or near Cambridge. In the spring of 1891 he caught a fresh cold, and since June, when he had a mild pleurisy, he had suffered from cough and morning expectoration, often streaked with blood. For some weeks past the expectoration had been more considerable, diffused, muco-purulent, and more or less stained with blood. Specimens of the sputum examined on October 15 and 17, 1891, were described as follows: "October 15: Purulent, blood-stained; no tubercle bacilli; few micro-organisms of any kind. In unstained specimens epithelial débris, small in amount, from buccal-respiratory tract. October 17: Two samples having the same character; purulent, blood-stained; no

tubercle bacilli; in unstained specimens pus cells, many undergoing fatty degeneration, also large lymph cells degenerated; large zooglœa groups of micrococci; very few epithelial elements."

The physical signs are shown in the annexed diagram (Fig. 35). Some fulness was observed on the right front of the chest from the second to the sixth rib-space; over this area, and extending laterally to the anterior axillary line and to the left margin of the sternum, the percussion note was dull in the centre, shading off at the margin, so as to present a rounded outline. Over the dull space the respiratory murmur was annulled, vocal fremitus diminished, and voice not conducted. Along the upper margin of dulness, in the subclavicular

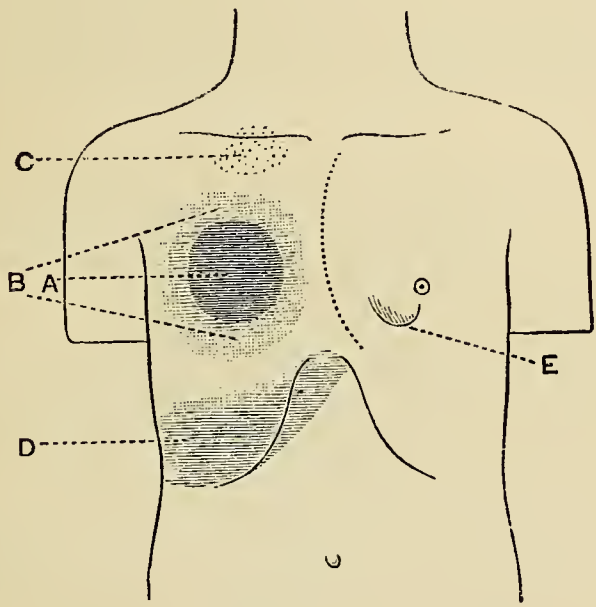


FIG. 35.

A, Centre of dense dulness and fulness of surface; B, area of lighter dulness, with annulled breath-sound and fremitus marking limits of tumour; C, shell of lung, giving high-pitched resonance, weak respiration, and a few fine râles; D, liver dulness, between which and area of tumour there is a narrow zone of resonant lung, giving weak but vesicular breath-sound; E, heart's apex-beat a little to left of the normal.

regions, the percussion note was high-pitched, tubular, and the respiratory murmur weak and accompanied by a few fine râles. At the lower border, between it and the liver dulness, there was also a narrow area of resonance and weak but vesicular breath-sound. The left lung was enlarged to include the left half of the sternum and covering the precordial region. The heart's apex-beat was slightly to the left of the normal point. Posteriorly, on the right side, the respiration was weakened over the upper half, with some fine congestion râles at the extreme apex, and some similar râles at the base; the percussion note in the interscapular region was slightly raised.

The diagnosis of hydatid of the lung having been made, aspiration,

at that time a recognised method of treatment, was advised, and this was performed between the fourth and fifth ribs by the late Dr. Lucas, of Cambridge, on November 4, one pint of clear hydatid fluid being removed by the instrument. Five minutes later another pint tinged with blood came with a rush from the throat, and for some days afterwards Mr. G. had a bad cough, spitting up cyst membrane and fœtid pus. The expectoration for some weeks amounted to a pint and a half each day, but gradually diminished to half a pint, then ceased.

When seen again on February 9, 1892, he had no cough, his general condition was good, and he was again on his way to St. Leonards. There was slight fulness and want of resonance in the right second space; otherwise nothing was notable beyond some weakness and harshness of breath-sound. Dr. Lucas, in a letter dated December 12, 1892, reported him as being in robust health.

Case 2.—Mr. X., aged twenty-three, was born in New Zealand and had lived there all his life until, in September, 1916, he left for England as a member of the New Zealand Expeditionary Force. He was a healthy, well-grown man, and had always enjoyed good health. He had previously been engaged in office work.

His illness commenced in March, 1917, when serving in France. The first symptom complained of was pain in the lower part of the left side, accompanied by slight pyrexia lasting for a few days. This was followed by cough with phlegm, which was sometimes streaked with blood. These symptoms continued, and on June 2 he had a sharp attack of hæmoptysis, and on June 13 a second attack, on this occasion bringing up several ounces of blood. Except for the slight and passing pyrexia referred to above there had been no rise of temperature. The sputum was examined for tubercle bacilli, but they were not found.

He was invalided home to England and admitted to a base hospital, when the diagnosis of pulmonary tuberculosis was made and sanatorium treatment recommended. He was, however, transferred to the New Zealand General Hospital at Walton-on-Thames, where the possibility of hydatid disease was recognized, in view of the frequent occurrence of this malady in the Dominion, and on being X-rayed the regular, well-defined shadow of the cyst was at once seen, thus confirming the diagnosis.

On June 29, through the courtesy of Colonel Myers and of the New Zealand Authorities, one of us had the opportunity of visiting the hospital at Walton-on-Thames, in which at the time were five cases of hydatid of the lung, and found the physical signs in the case of Mr. X. to be as follows: On the front of the chest percussion and auscultation were natural. On the back a somewhat oval area of dulness was present, involving the lower scapular and interscapular region on the left side, extending from the fourth to the eighth rib posteriorly, as indicated at A, Fig. 36. For some distance around this the note was flattened (B, Fig. 36), but below this to the bottom of

the chest the percussion note was resonant. Over the dull area the vesicular murmur was but feebly heard, and vocal vibration was diminished. No bronchial breathing or moist sounds were audible. On examination the patient with the X-ray screen, a well-defined shadow the size of a cocoanut, with regular outline, having all the appearance of a hydatid cyst, was clearly visible. From its better definition when viewed from behind, it was clear that the cyst was nearer the posterior than the anterior wall of the chest, thus agreeing with the physical signs.

The diagnosis having been made, it was decided to treat the case by resection of rib and drainage, as described in a further paragraph (see p. 377). The operation was successfully performed on June 30, and on August 8 Mr. X. was evacuated home to New Zealand, convalescence being somewhat retarded by infection of the wound, which manifested itself a week after the operation.

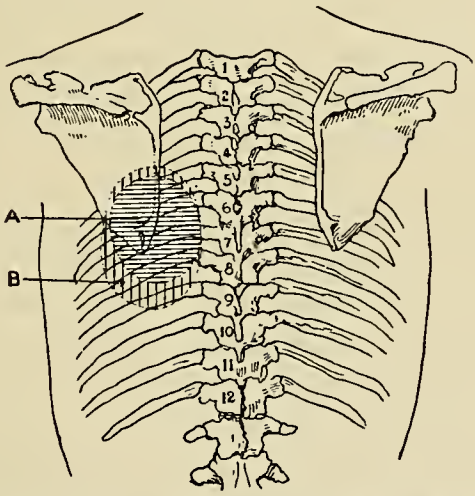


FIG. 36.—DIAGRAM SHOWING THE PHYSICAL SIGNS OF HYDATID OF THE LUNG AS MET WITH IN MR. X., AGED TWENTY-THREE, WHOSE CASE IS DESCRIBED IN THE TEXT.

2. *Rupture of the Cyst.*—Spontaneous rupture of the cyst into the bronchial tubes takes place in about half the cases, into the pleura in 5 per cent., and more rarely into the pericardium, pulmonary vein (Wilson Fox), or through the diaphragm (Dupuytren). It must be remembered also that an hydatid cyst of the liver may rupture upwards and the contents be expectorated.

Sudden pain, intense dyspnœa, and the expectoration of a large amount of watery blood-stained fluid, often having a peculiar and unpleasant taste, are the symptoms which immediately attend upon rupture of the sac into the lung. In some cases the lungs are so completely flooded as to overwhelm the patient and cause immediate death. In other cases the churn-

ing and rattling of the fluid and air in the chest can be heard, and after a desperate struggle the bronchial tubes become sufficiently cleared, when rapid amendment of symptoms takes place. Together with the fluid a greater or smaller number of daughter cysts may be expelled, and sometimes immediately, but more often at a subsequent date, the cyst wall may be expectorated entire, or more commonly in fragments, with symptoms of threatened suffocation. In some cases death has resulted from impaction of a portion of the cyst wall in the glottis. It frequently happens that with these alarming symptoms the real nature of the disease is for the first time disclosed, the expectoration of the hydatid membrane or daughter cysts being especially characteristic.

Hooklets and scolices are frequently to be found in the expectorated fluid on microscopical examination, and fragments of the cyst membrane reveal also the peculiar lamination so characteristic of hydatid, which remains unchanged long after the death of the parasite.

Hæmoptysis, much more profuse in quantity than in the earlier stages of the disease, may occur at the time of rupture, or at a subsequent period. It is due to the tearing away of the cyst membrane from its vascular capsule, from which, at the moment of collapse, the support and even pressure of the distended cyst is suddenly removed. This intracystic pressure was ascertained by Dr. Thomas [1b] in several cases of hydatid cyst to measure from 10 to 12 inches of water, and in one case, in which the cyst was large enough to bulge, and was therefore compressed by the thoracic wall, it amounted to 25 inches during inspiration, 30 inches during expiration. Hæmoptysis is thus one of the most constant and important symptoms of hydatid of the lung, occurring at some period in about four-fifths of the cases. As a rule only slight in degree at the earlier stages, it is often severe and repeated, sometimes even fatal, after the rupture of the sac.

Course of the Disease.—From time to time, as we have indicated, small hydatid cysts are unexpectedly met with in the lung after death, shrivelled and infiltrated with salts, and with their fluid contents partially absorbed and inspissated. But such instances of the death of the parasite, though not infrequent in the liver, are rare in the lung, and only occur in cysts of very small dimensions, which have probably given rise to

no symptoms. Dr. Lendon,[36] indeed, goes so far as to say that there is "no case on record in which a cyst diagnosed as such during life has been left alone, and has been found after the death of the patient to have undergone retrograde change." In considering treatment, therefore, such a happy event must not be anticipated. Hydatid cysts, indeed, large enough to give rise to symptoms and physical signs, and to be diagnosed as such, tend to enlarge, and finally to rupture, exposing the patients in that event to the danger of being suffocated by the sudden flooding of the bronchial tubes.

Of the large proportion of cases in which death is not caused by suffocation at the time of rupture, many recover; the cyst wall is gradually expectorated, and, the surrounding lung expanding, the cavity becomes closed. In other cases, after a longer or shorter interval, suppuration of the capsule takes place, and a pulmonary abscess, sometimes of a very fœtid type, results.

The emaciation, hectic, occasional hæmoptysis, severe cough, and often profuse expectoration that characterise the later stages of hydatid disease, in combination with the physical signs of excavation of the lung and of great bronchial irritation, especially on the side affected by the parasite, are at first sight suggestive of advanced pulmonary tuberculosis. A careful examination into the history of the case, however, including the sudden commencement of expectoration as a copious outburst of blood-stained watery or semi-purulent fluid, having a peculiar and unpleasant taste, and the discovery of the physical signs of a cavity in some situation unusual in phthisis, will arouse suspicions as to the true nature of the disease. A careful daily observation of the sputum, which is, moreover, often fœtid, may lead to the discovery of membranous shreds or the more typical "gooseberry skins," of which microscopic examination will reveal the structure to be that of hydatid cyst.

Diagnosis.—From the early occurrence of hæmoptysis and cough hydatid disease of the lung may be mistaken, as we have seen, for phthisis. If situated in the lower part of the lung it may simulate a pleuritic effusion, and the more so as some degree of pleurisy with slight effusion not uncommonly accompanies the cyst-formation, though not, as a rule, sufficient to obscure the X-ray picture.

The possibility of hydatid affection should be borne in mind in patients coming from Australia and New Zealand or any country in which the malady is common, and an X-ray examination of the chest should be made in all doubtful cases.

In the later stages of the disease, when suppuration has occurred, the case has to be discriminated from advanced phthisis, suppuration of the lung, and other allied conditions. A repeated examination of the sputum is then of great value.

Treatment.—A review of the statistics of cases left untreated would place the natural mortality of the complaint at between 50 and 60 per cent.[5] The disease cannot, therefore, be left alone, and medicinal remedies being of little use, recourse must be had to more active methods of treatment.

Until recently, paracentesis of the cyst and the evacuation of as much of its fluid contents as possible, thus causing the death of the parasite, was the method usually employed. Under this line of treatment certain cases no doubt recovered which under the expectant treatment would have died, but in some cases death has directly followed the operation[6] (Plate XXI.). This arises in certain instances from the trocar becoming blocked by a portion of the cyst wall or by a daughter cyst, and failing therefore to extract more than a few ounces of fluid; when the instrument is withdrawn, the elastic cyst retracts, and the contents are discharged into the cavity in which it lies, thus flooding the bronchi and leading to suffocation, as in spontaneous rupture.* In other cases it is probable that the paracentesis produces not merely a fine hole in the cyst wall, but a tear in the chitinous envelope, thus more easily leading to the escape of the contained fluid.

For the above reasons paracentesis can no longer be recommended, and we should in all cases in which the cyst is accessible proceed at once to the radical operation of incision and removal, which constitutes the proper treatment of such cases. For similar reasons, exploratory puncture for diagnostic purposes, even with the finest needle, should not be countenanced until resection of a rib has been performed and the surgeon is ready at once to incise the cyst. Moreover,

* It is interesting to note that the blocking of the trocar by a cyst during paracentesis was a condition well known to ancient writers (see Aretæus, *On the Causes and Symptoms of Chronic Diseases*, book ii., chap. i., p. 337, Sydenham Society edition).

PLATE XXI

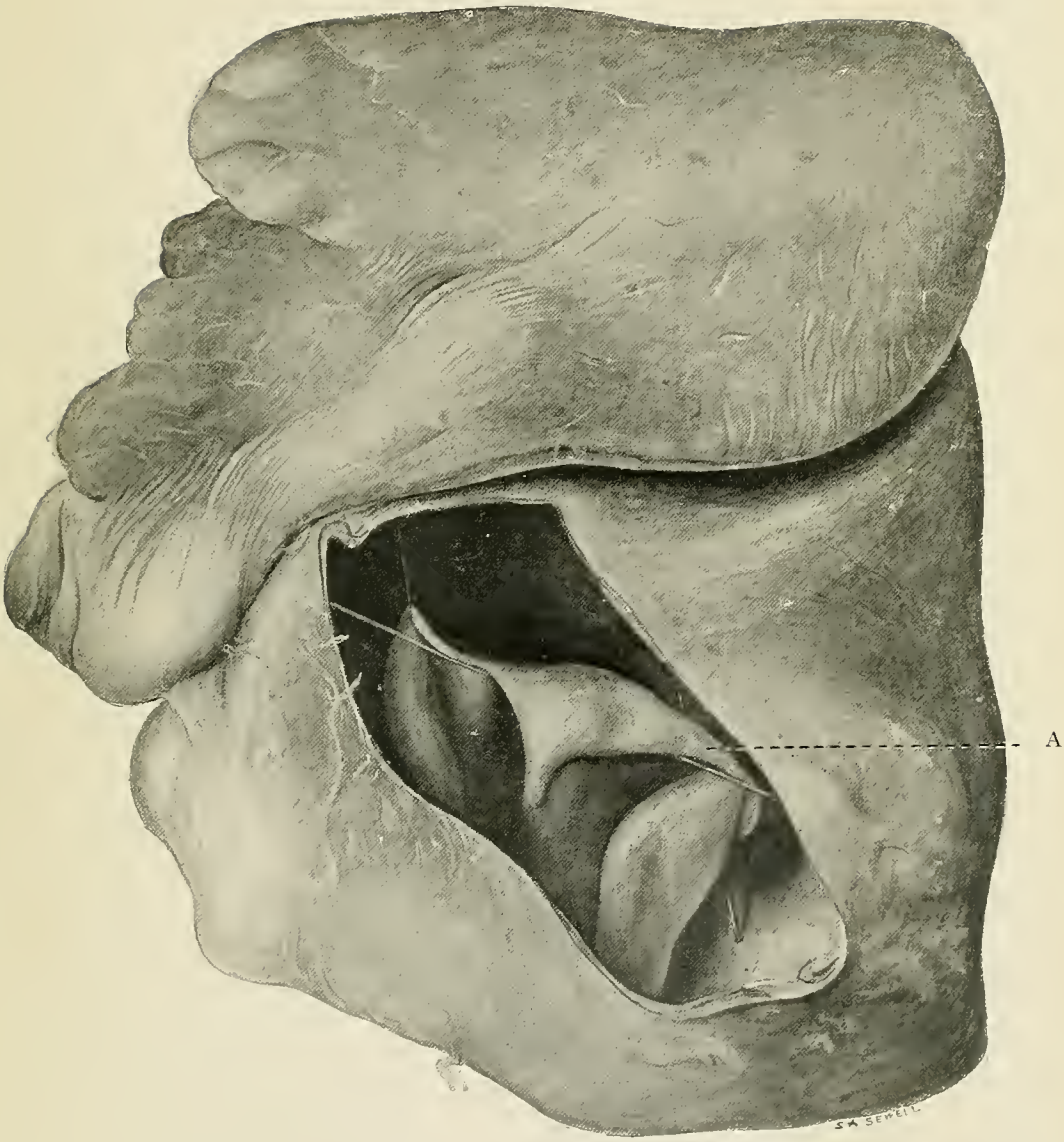
HYDATID OF THE LUNG

The drawing shows the left lung of a boy aged nine years. In the lower lobe is seen a large cavity about three inches in diameter, in which lies collapsed and coiled up a solitary hydatid cyst (A). From the cavity a communication with the left bronchus had been effected by an ulcerated aperture, through which a bristle has been passed. By this means the fluid within the hydatid made its way into the bronchial tract after puncture of the cyst, and caused death by suffocation. The pleura over the outer aspect of the cyst is covered with recent lymph.

From a patient who presented symptoms and signs suggestive of pleurisy with effusion. Paracentesis was performed, but only a few drachms of clear watery fluid were evacuated. The boy almost immediately began to cough, and brought up 3 or 4 ounces of clear frothy fluid. He died of suffocation within eight minutes of the paracentesis. The case is recorded by the late Dr. Bristowe in the *Transactions of the Clinical Society*, 1891, vol. xxiv., p. 73.

(From the Museum of St. Thomas's Hospital. ¾ natural size.)

PLATE XXI



HYDATID OF THE LUNG.

To face p. 376.

since the introduction of the X-rays exploratory puncture has become unnecessary, for hydatid cysts present, as we have said, a picture which is strongly suggestive of the disease.

Operation having been decided upon, resection of a rib must be performed and the pleural layers united by suture. The cyst must be then boldly and freely opened with the aid of expanding forceps, and its fluid contents allowed to escape quickly, to avoid the flooding of the lung which might otherwise result. To assist the evacuation, it is wise to turn the patient well over on to the affected side at the moment of incising the cyst. The chitinous envelope can as a rule be easily detached from the surrounding lung and the cyst withdrawn. The wall of the cavity should then be swabbed with a 1 per cent. solution of formalin to render it aseptic, and thus prevent post-operative recurrence from the growth of an escaped scolex.[7] The cavity and wound in the chest-wall may then be closed, but should it prove impossible to evacuate the cyst, or should suppuration have occurred, drainage must be undertaken. The results of this method of treatment are very satisfactory. To quote only those of more recent date, we find that of 99 cases reported upon by Garré and Quincke,[8] 78 recovered and 21 died, a mortality of 21·2 per cent. Guimbellot,[9] collecting cases from the literature, found that among 223 patients treated by thoracotomy, 194 were cured and 29 died, a death-rate of 13 per cent. Dividing the cases into those in which the cyst was healthy at the time of the operation, and those in which suppuration had occurred, the statistics yielded a mortality of 8·2 per cent. in the case of the former, and of 19 per cent. in the latter. These figures contrast favourably with the death-rate of 55 per cent. among patients left untreated. The value of radical surgical treatment as indicated by the earlier statistics of Dr. Thomas[2b] is thus amply demonstrated.

Should spontaneous rupture occur, each case must be treated on its merits. As we have seen, many such patients eventually recover, gradually coughing up the membrane and evacuating the cyst. But if suppuration has set in, and if, in spite of a generous supporting diet and the administration of such remedies as bark and mineral acids, the patient is evidently losing ground from hectic and profuse expectoration, it will be wise to treat the case surgically, and to open and drain the

cavity on the lines already indicated for abscess of the lung (see p. 361).

If the hydatid cyst be in the pleural cavity, a condition of rare occurrence, it will probably be mistaken for a pleural effusion until exploratory puncture reveals the true nature of the case. Resection of a rib and evacuation of the cyst is then the proper method of treatment.

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[1] (*a*) *Hydatid Disease,* by John Davies Thomas, M.D., F.R.C.S., p. 122. Adelaide, 1884.
(*b*) *Loc. cit.,* p. 21.

[2] (*a*) *Hydatid Disease,* by the late John Davies Thomas, M.D., F.R.C.S., edited by Alfred Austin Lendon, M.D., vol. ii., p. 38, Sydney, 1894.
(*b*) *Loc. cit.,* vol. ii., pp. 142, 160, 161.

[3] (*a*) See *Clinical Lectures on Hydatid Disease of the Lungs,* by Alfred Austin Lendon, M.D., p. 116. London, 1902.
(*b*) *Loc. cit.,* p. 23.

[4] (*a*) See L. C. Zapélloni (*Il Policlinico,* Rome, 1915, sez. Chirurg., xxii.), *British Medical Journal,* 1916, i., Epit. No. 29.
(*b*) *Infection, Immunity, and Specific Therapy,* by John A. Kolmer, M.D., D.Ph., M.Sc., p. 524. Philadelphia and London, 1917.

[5] *The Surgery of the Chest,* by Stephen Paget, M.A., F.R.C.S., p. 411. Bristol and London, 1896.

[6] See, amongst others:

(1) "Case of Living Hydatid of the Lung, in which Aspiration was followed immediately by Subcutaneous Emphysema and by Suffocation due to the Rush of Hydatid Fluid into the Bronchial Tubes," by J. S. Bristowe, M.D., F.R.S., *Transactions of the Clinical Society of London,* 1891, vol. xxiv., p. 73.

(2) A "Case of Hydatid of Lung which proved Fatal by Rupture into a Bronchus Nine Hours after Treatment by Aspiration," by Hector W. G. Mackenzie, M.D., *Transactions of the Clinical Society of London,* 1892, vol. xxv., p. 215.

[7] "Observations on the Treatment of Hydatid Disease from the Points of View of (1) Prophylaxis, (2) Aspiration or Tapping, and (3) the No-Drain Operation of Bond and Others," by L. E. Barnett, F.R.C.S., Professor of Surgery, University of Otago, *New Zealand Medical Journal,* May, 1914, p. 145.

[8] *Surgery of the Lung,* by C. Garré and H. Quincke, second edition. Translated from the German by David M. Barcroft, M.D., p. 214. London, 1912.

[9] *Sur le Traitement Chirurgical des Kystes Hydatiques de la Plevre et du Poumon.* Thèse pour le Doctorat en Médecine, par Marcel Guimbellot, p. 40. Paris, 1910.

CHAPTER XXIV

INTRATHORACIC DERMOID TUMOURS

Sir John Bland-Sutton[1] classifies those dermoid tumours as "sequestration dermoids," which arise in detached or sequestrated portions of surface epithelium. They occur chiefly in situations where, during embryonic life, coalescence takes place between skin-covered parts, namely, along the median line, in front or behind. The thoracic dermoids of this type occur in two situations:—

(1) On the anterior aspect of the sternum, usually in the middle line, about the junction of the first and second sternal pieces, where they present as external tumours.

(2) In the thoracic cavity.

Sir John Bland-Sutton would regard such *intrathoracic dermoids* as due to the sequestration of a piece of skin during the development and junction of the two halves of the sternum, and the subsequent dislocation backwards of the involved portion to the deep surface of the bone. Here it may remain indefinitely dormant, or become active and gradually enlarge to form a cystic tumour. Such cysts are probably always mediastinal in their commencement, and may so remain throughout their course. In other cases the tumours rapidly enlarge, extending their boundaries, not passively by mere distension from within of accumulating contents, but actively, so that the term dermoid *growth*, rather than dermoid *cyst*, should be applied to them. As they enlarge they are apt to become closely adherent to the pericardium and pleura and to penetrate deeply into the lung, where they become so embedded that their original connection with the mediastinum is obscured. A communication with the lung or a bronchus may at any time be established, and on the accession of air into the cavity suppuration is liable to ensue.

On section, such tumours are found to contain sebaceous material, and often hairs. In other cases the cyst contains in

addition such structures as muscle, cartilage, bone or teeth, and would then belong pathologically to a different category, and should be classified among the *teratomata.*

Intrathoracic dermoids, including teratomata, are very rare, and Dr. Batty Shaw and Dr. Williams,[2a] in their paper dealing with this subject, were only able to find records of thirty-five authentic cases, a total recently brought up to fifty-two by Dr. Pohl.[3] From an analysis of these cases the following facts appear.

The sexes are affected equally. In some of the cases the tumour has remained of small size and has been only found by accident after death. In others *symptoms* have supervened which caused the patient to seek advice. The age at which such symptoms manifest themselves varies, being sometimes as early as sixteen, though "in by far the larger number the age was between twenty and thirty years." Careful cross-questioning may, however, elicit the fact that hairs have been expectorated for some years previously; this had occurred in one case on many occasions since the patient was sixteen years of age, and in another at fifteen, the earliest age at which it has been recorded. This symptom, which was present in seven of the thirty-five cases referred to above, is a most characteristic feature of the disease, and should always be carefully inquired for. It is of importance also as showing that a communication exists between the interior of the cyst and the air-passages, with the danger of suppuration which such a communication entails.

Other symptoms, such as cough, expectoration, shortness of breath, and sometimes hæmoptysis, are produced by the enlargement and irritating effects of the cyst, which not uncommonly reaches the size of a child's head, or even larger. The temperature is as a rule but little raised, unless suppuration or septic pneumonia ensue. Fever, night-sweating, and wasting then supervene. In some cases, as in that to be immediately related, an empyema occurs, and may entirely mask any characteristic signs.

The *physical signs*—impairment of note over the tumour, with weak or absent breath-sounds, diminished vocal resonance and vocal vibrations—offer nothing characteristic. It is stated that compression of veins is much less common than in cases of malignant growth in the mediastinum, a point of some

diagnostic importance. It occurred, however, in a marked degree in a case reported by Dr. Mouat.[4]

The X-rays, provided the surrounding lung and pleura be still healthy, reveal a somewhat rounded shadow, with margin well defined, except perhaps on the inner side, and without pulsation. Such a picture might lead the observer to consider the possibility of a dermoid cyst, but in the absence of the expectoration of hairs, or of an exploratory operation, the exact diagnosis must remain a matter of uncertainty.

The disease, if untreated, marches slowly to a fatal termination, more than half the patients dying before the age of thirty. The end is brought about by suppuration, septic pneumonia, pulmonary tuberculosis, and sometimes with symptoms of pressure; in other cases the cysts have been known to rupture into the pericardium and the superior vena cava. The average age at death in seventeen cases collected by Drs. Batty Shaw and Williams[2a] was thirty-two, the youngest patient being twenty, the oldest sixty.

Drugs are of no avail in the treatment of this disease, and surgical intervention must be considered. Of seven cases thus treated, quoted by the above authors, in one, in which the cyst was small, complete enucleation, with recovery of the patient, was effected. But such a result is exceptional. In most cases, before the cyst has given rise to decided symptoms, and thus come under observation, it has already attained considerable dimensions, and has formed attachments to the pericardium, lungs, pleura, or diaphragm, so that its complete removal is impossible. For a similar reason complete retraction of the cyst walls after operation is unlikely. Nevertheless, great diminution in size of the cavity has followed incision and drainage in most of the cases operated upon, the patient being left with a small fistula only. In one case alone, and that the earliest, was the result unsatisfactory.

In view, therefore, of the steady progress of the disease to a fatal termination, if not actively treated, the question of surgical intervention should always be carefully considered.

The details of the following case, sent to Dr. Douglas Powell by the late Dr. Sturges, of Beckenham, and seen by him six years later with the late Sir Lauder Brunton and Sir Rickman Godlee, will illustrate the more important features of the

disease, although, as we shall presently point out, in view of later investigations there is some room for doubt whether its real origin may not have been teratomatous. In this instance it will be noticed that but little retraction of the cavity took place after operation.

At the first consultation in 1881, the lady, aged twenty-nine, presented all the signs and symptoms of a purulent effusion into the right pleura, which had supervened upon an acute right pleurisy the preceding May. There were no physical signs to differentiate the case from an ordinary empyema, the presence of pus being inferred from the prolonged duration and hectic character of the symptoms. Exploration, with a view to thoracentesis, was advised, but was deferred, as the lady was on her way to the South of France. The operation was then still further postponed, and, a few months later, rupture through a bronchus took place, and a large quantity of matter was expectorated. The empyemic cavity partially contracted, the expectoration gradually diminished, and the lady regained strength and flesh, and subsequently married.

Mrs. W. was not seen again by Dr. Powell until six years later, in June, 1887, when Sir Lauder Brunton consulted him about her, she having somewhat failed of late, losing flesh, showing a daily, although slight, rise of temperature, and presenting a trace of albumin in the urine. The side was now considerably contracted, the ribs approximated, and the signs of the empyemic cavity occupied the antero-lateral region of the lower right chest, extending also somewhat backwards, where it was bounded above by a downward slanting line skirting the angle of the scapula. There was still nothing about the case to suggest any other diagnosis than that of an old empyema cavity communicating with the lung through a bronchial fistula. It was agreed that the cavity should be drained from without, and, on June 18, this was effected by Sir Rickman Godlee, who excised a portion of the sixth rib in the anterior axillary line, having previously punctured at this point, where there was some tenderness, and found pus. About an ounce of pus escaped, and some caseous, putty-like-looking stuff was removed. The patient again improved, but the discharge from the wound continued.

On August 9 the cavity was again explored. At its orifice a hair was noticed, and on introducing a small sponge into the cavity a number of hairs were found to adhere to it, and subsequently a handful of matted hairs and putty-like débris was removed. The nature of the case was now for the first time apparent, and the patient, on being questioned, stated that she had coughed up two hairs since the previous operation, and also on one or two occasions long previously, and that her brother, who had been dressing the wound, had observed one or two on the dressings. On carefully exploring the cavity in the light of this new discovery, its irregular interior was found to be due to finger-like processes consisting of

excrescences of fibro-cellular tissue covered with skin, from which the hairs were growing. At a subsequent operation in September the opening was enlarged by excising a portion of a third rib, and one of these processes was removed close to its base by Paquelin's cautery, and other small ones ligatured and removed by scissors.[5] It was found that the cavity extended up to the apex of the thorax, and that the wall of the cyst was intimately connected with the surfaces of the lung and diaphragm. The internal parts were somewhat freely cauterised, and the skin of the thorax was then sutured to that of the dermoid cyst, and the cavity loosely plugged with boracic lint. Under this skilful handling by the surgeon the patient rapidly improved, although discharge remained and required daily irrigation. The patient held her own, wintering at San Remo for two or three years, and finally died from drain poisoning at some mountain resort in the summer of 1891.

In this case, in the absence of post-mortem examination, we may infer that the malady started in the upper mediastinum by extension from a remnant of dermal tissue involuted and detached, and for a time embedded in the tissues. The first effect of its active growth and intrusion into the pleural cavity was to set up pleurisy and empyema, the dermoid growth gradually extending to occupy much of the cavity. The empyema, with which it was complicated, then perforated the pulmonary pleura and thus permitted some of the hairs from the growth to escape through the lungs.

A case of "dermoid growth of the lung" closely resembling that above described was brought before the Pathological Society by Dr. Cyril Ogle,[6] in which the cyst was embedded in the lower lobe of the left lung, occupying a cavity of about four inches in diameter, and communicating freely with the left bronchus. The contents were of the usual character, including five or six stalked, tongue-like processes with short hairs growing upon their surfaces. The stalks were united and appeared to grow from the wall of the cyst, but the united stem could be traced upwards into the mediastinum at the level of the left innominate vein. Embedded in the common stem or core a large tooth was found, and this fact places the case amongst those of teratomata. The specimen was taken from a man, aged twenty-eight, who had died of hæmoptysis.

Although it may be regarded as in the highest degree probable that some intrathoracic dermoid growths have their origin in "the inclusions of portions of the primitive epiblast during the closure of embryonic clefts" (Shattock[7a]), it is

likely that a further and more complete examination of such cases as that of Mrs. W., above related, may prove that the majority of them are really teratomata, abortive embryos, whose origin is explained on the theory of embryo-genesis put forward by Mr. Shattock.[7b] The structural resemblances between the cyst described by Dr. Cyril Ogle and the one which we have above related are great. It was only on close post-mortem examination in Dr. Ogle's case of the stalk whence the fleshy, hair-covered processes projected that a tooth was found. In our case the patient died of another malady, no autopsy was made, and the only structures examined were some of the fleshy processes removed by Sir Rickman Godlee.

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(*b*) "Case of Mediastinal Dermoid," by G. E. O. Williams (introduced by Batty Shaw, M.D.), *Transactions of the Clinical Society of London,* 1906, vol. xxxix., p. 210.

[3] The following papers give a nearly complete record of the cases published up to the year 1914:

(*a*) "Beiträge zur Genese. Pathologie und Diagnose der Dermoidcysten und Teratome im Mediastinum anticum," von Dr. Bruno Dangschat, *Beiträge zur Klinischen Chirurgie,* vol. cxxx., 1903, p. 692.

(*b*) "Über Mediastinal-Dermoide," von Dr. W. Pohl, *Deutsche Zeitschrift für Chirurgie,* vol. cxxx., 1914, p. 481.

[4] "Case of Suppurating Dermoid of the Mediastinum," by Thomas R. Mouat, M.B., *British Medical Journal,* 1909, vol. i., p. 90.

[5] See Sir Rickman Godlee's paper dealing with the case—"Dermoid Cyst of the Right Side of the Chest communicating with a Bronchus," by Rickman J. Godlee, M.S., *Transactions of the Royal Medical and Chirurgical Society,* 1889, vol. lxxii., p. 317—in which the curious processes excised are figured.

[6] "Dermoid Growth of the Lung," by Cyril Ogle, M.D., *Transactions of the Pathological Society of London,* 1897, vol. xlvii., p. 37.

[7] (*a*) "An Acardiac Acephalous Ovarian Embryoma, with Remarks on the Pathogenesis of the so-called Dermoid Cyst of the Ovary," by S. G. Shattock, *Transactions of the Pathological Society of London,* 1907, vol. lviii., p. 273.

(*b*) *Loc. cit.,* p. 303.

CHAPTER XXV

SYPHILITIC DISEASE OF THE BRONCHI AND LUNGS

Bronchial Syphilis.

THE respiratory organs may become affected in the secondary or tertiary periods of syphilis. In the eruptions of secondary syphilis the bronchial mucous membrane is sometimes involved. It is true that the positive evidence of macular syphilis of the bronchial tubes is incomplete, and not confirmed by post-mortem observation; but very decided symptoms of bronchial catarrh are so frequently met with in association with the secondary cutaneous rash of syphilis and the corresponding throat affections that it is impossible to escape the conviction that a specific catarrh of the respiratory tract is present in such cases. Moreover, condylomata may occasionally be visible in the larynx. The catarrhal symptoms and mottled cutaneous eruptions of syphilis, together with a certain degree of pyrexia, may be mistaken for those of measles.

We have not observed asthma in association with the bronchial eruption in syphilis.

Syphilitic ulceration of the bronchial tubes is of rare occurrence during the secondary period of the disease, and is only occasionally to be met with in association with tertiary lesions. When a main bronchus or the bifurcation of the trachea is the seat of a syphilitic ulcer, paroxysmal cough and dyspnœa, with scanty expectoration occasionally streaked with blood, may be expected, and the diagnosis would be helped by the observation of other phenomena of syphilis. The ulcer may perforate the trachea or bronchus, and produce mediastinal or pulmonary abscess; or a branch of the bronchial artery may be opened up, leading to severe or fatal hæmorrhage. But the tendency of the syphilitic ulcer is, after deep erosion of the tissues, to cicatrise, causing contraction and

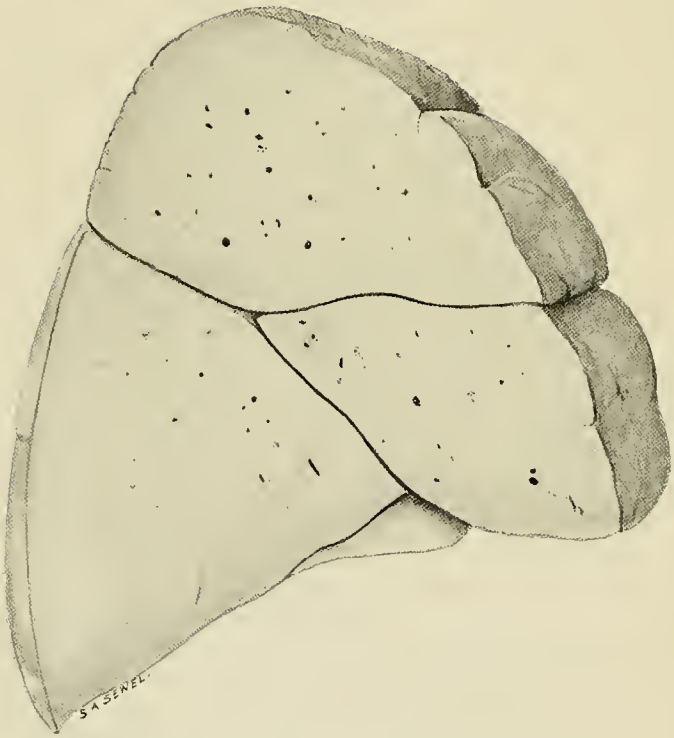
deformity of the tubes affected, and leading later to bronchiectasis and fibrosis of the lung. We have already alluded to narrowing of the bronchi as one of the dreaded consequences of syphilis (see p. 205). Syphilitic ulceration of the smaller tubes is of occasional occurrence, and may extend into the lung, producing peribronchial pneumonic consolidations.

The clinical recognition of bronchial syphilis rests upon (1) the presence of symptoms of broncho-pulmonary disease which are not in the order of those characteristic of simple bronchitis or phthisis; (2) the history of the patient and the manifestation of the syphilitic cachexia in other directions; (3) the rapid amendment, provided the disease be not too far advanced, that ensues upon antisyphilitic treatment.

Pulmonary Syphilis.

Syphilitic lesions are, however, not confined to the bronchi, but may occur in the lungs themselves, both in the congenital and acquired forms of the disease. In the days anterior to the discovery of the tubercle bacillus this affection was believed to be of common occurrence; but it is now known that many of the supposed cases were in reality tuberculous in nature, and that the disease is of some rarity. Sir J. Kingston Fowler,[1] after a careful inspection of the museums of the London hospitals and of the Royal College of Surgeons, was unable to find more than "twelve specimens which are believed to illustrate syphilitic lesions of the lungs," and of these he excluded two as very doubtful examples. This museum experience, however, would only in a measure coincide with the clinical experience of the disease, since cases recognised will often yield to appropriate treatment. On referring back to clinical notes of 1,323 male patients suffering from various diseases, none of them hospital cases, and of the age of twenty and upwards, seen by one of us, 53 cases presented a definite history of syphilis; out of this number there were 5 cases of lung syphilis—viz., 3 cases of gummatous fibrosis, one of catarrh associated with cutaneous maculæ, and one of acute pyrexial lung syphilis, described later in the text (p. 389). On the other hand, out of the same series of cases there were 320 cases of pulmonary tuberculosis, in which only in 3, or less than 1 per cent., was a syphilitic history apparent.

PLATE XXII



Congenital Syphilitic Pneumonia.

To face p. 387.

CONGENITAL SYPHILITIC PNEUMONIA

The illustration shows "a right lung affected with syphilitic pneumonia. All the lobes are equally affected, the lung tissue presenting a white hepatised appearance. There is no evidence of any recent inflammation of the pleura, nor any adhesion of the lobes to each other. The surface of the lung is rough and nodular.

"Microscopical examination revealed a generalised interstitial inflammation with marked thickening of the adventitial coat of the arteries. A few of the alveolar spaces have expanded, and contain numerous desquamated cells."

From an infant who only lived for a quarter of an hour. A bullous eruption was present on its body, and the hands and feet were partly denuded of epithelium. The lungs were entirely solid, and the liver and spleen were enlarged.

(From the Museum of St. Bartholomew's Hospital.
Natural size.)

PLATE XXII

Congenital Syphilis.—In this form of the disease gummatous nodules are occasionally met with; but more often the lesion is found to take on a more diffused form, producing the so-called "white pneumonia," or "white hepatisation," of Virchow, as seen in the syphilitic fœtus or new-born child. A lung affected with syphilitic pneumonia is heavy and solid, of greyish-white colour, and smooth on section (Plate XXII.), thus presenting a very different appearance from the granular, friable surface seen in ordinary pneumonia. Microscopically, the alveolar walls are found thickened and infiltrated, showing diffuse chronic interstitial inflammation. The adventitia of the arteries is thickened, and the alveoli are filled with shed epithelium and round cells, often in a state of fatty degeneration.[2] With proper staining, spirochætes, often in very large numbers, may be demonstrated in such lungs.

Congenital syphilitic lung disease in infants is a matter more frequently of post-mortem than of clinical observation, the cases in which it occurs being generally fatal within a very few hours of birth; hence it is of small clinical importance. No doubt congenital syphilis has something to do with catarrhal and even tuberculous lesions which develope in later childhood and adult life, but the measure of its influence in such affections cannot be definitely traced.

Acquired Syphilis.—The lung is attacked by this form of syphilis generally in from four to six years or more after the primary infection, but we have known twelve years to elapse before its manifestation. The diffused form of the disease, seen in the fœtus and in new-born children, is rarely, if ever, met with, gummata or their results being the common manifestations. These may occur in any part of the lung, this irregularity of site being a distinctive feature. In appearance they resemble gummata in other parts, and, as in the liver, present at first a rose-grey appearance. Unlike the tuberculous nodule, which is non-vascular, the gumma contains vessels, and its nutrition is thus for a time assured. Gradually, however, the vessels undergo the changes known as endarteritis obliterans, the blood-supply becomes cut off, and the fibro-cellular elements, of which the gumma is composed, undergo caseation. In this way are produced the yellow caseous masses, surrounded by the fibrous capsule, with which we are more familiar. Softening and breaking down occasion-

ally ensue, but the cavities so formed, unlike their tuberculous counterparts, are rarely large. More often the gummatous mass becomes converted into fibrous tissue, which, radiating into the lung around, causes puckering and scarring of the viscus.

There can be little doubt that cases occur, perhaps with greater frequency than is suspected, in which syphilis of the lung, with destructive changes, softening and excavation, associated with hectic phenomena and wasting, remains untreated, being from the first mistaken for tuberculous disease. We must admit, however, that the majority of cases believed by older writers to be of this nature were in reality instances of true pulmonary tuberculosis occurring in syphilitic subjects, while a few, as in a good example to be seen in the Brompton Hospital Museum,[3] are the result of syphilitic stenosis of the trachea or bronchi, leading (like other varieties of stenosis) to the formation of bronchiectatic cavities and cirrhosis of the lung.

Symptoms.—The symptoms of pulmonary syphilis are often obscure, and may readily be confounded with those of ordinary inflammatory conditions. Gummatous nodules have been found in those who during life presented no evidence of pulmonary disease. The symptoms generally present are those of chronic indurative disease of the lung or pleura — a paroxysmal cough with difficult expectoration, of obstinate continuance uninfluenced by ordinary remedies, and associated with but little emaciation and no pyrexia. Pleuritic pains are often present. The presence of such symptoms in a person bearing the marks or giving a history of syphilis would be very significant. It not infrequently happens, however, that some peculiarity or incongruousness in the symptoms or physical signs leads to the suspicion of a syphilitic taint, the surface marks of which are obscure, and the history of which is at first denied, either intentionally or through ignorance. In such cases a Wassermann's test of the patient's blood may prove of value.

Hæmoptysis is rare in the early stages, and when it occurs is highly suggestive of tuberculous complication. Cases, moreover, do occur, such as one to be presently related, in which more acute symptoms present themselves, including a remittent pyrexia, and such cases very closely resemble tuberculosis.

Physical Signs.—The most characteristic physical signs are those of localised pulmonary induration—flattening, dulness, or a sense of hardness on percussion, with enfeebled breath-sound of blowing quality, with few or no moist sounds. Some bronchial clicks, or, more commonly, a superficial crackle of pleuritic or subpleural source may be heard. Such signs, when presented at some unusual situation, as about the mammary or inframammary or infraspinous region, are very suggestive. It is certain, however, that in some cases the physical signs are not localised in any unusual spot, but present themselves at one or other apex, and under these circumstances the diagnosis from chronic tuberculosis is most difficult. The presence of considerable one-sided indurative disease of the lung, of chronic course and not traceable to any preceding acute attack, in a person who has not been engaged in any dusty employment, and whose sputum contains no tubercle bacilli, should lead to a suspicion of syphilis. Having arrived thus far, a careful inquiry into the history, an examination for surface marks of the disease, and a Wassermann test, will usually clear up the case. The sputum should also be examined for spirochætes.

Syphilitic gummata, as we have said, sometimes soften and produce cavities, which are small, however, and often too deeply seated for recognition. In such cases severe hæmoptysis may occur from the rupture of a large vessel.

Should the larynx be involved, an examination will disclose the cleanly cut and widely destructive ulceration of syphilis, or in later and less active stages the evidence of scarring and deforming cicatricial changes.

The following case illustrates the great similarity which may hold between syphilis and tuberculosis:

Mr. P., aged thirty-one, engaged in commerce, was seen by one of us in consultation with Dr. Bulger, of Holloway, on August 4 and 5, 1909. He admitted of no illness until he began to feel unwell and to cough in the beginning of January, 1909. His cough continued, he lost flesh, and at Easter there was some staining of the expectoration. The cough became more severe in April, and since the middle of June he had been in bed on account of increasing weakness and a daily rise of temperature. The expectoration was examined in May with a negative result as regards tubercle bacilli. The opsonic index for tubercle was found to be 0·62. He was thin and hectic-looking; his teeth and gums were in a bad condition, and pyorrhœa alveolaris was present.

On careful examination an area of dulness in the right scapular and interscapular region was discovered, extending from the second to the sixth rib. Over this area the breath-sounds were feeble, and some scattered moist crepitations were audible. These signs, with the symptoms, were regarded as indicative of pulmonary tuberculosis, although it was recognised that the duration of the case, the comparative paucity of the physical signs, and the absence of tubercle bacilli, confirmed by a further examination of the sputum, were anomalous features. The question was duly considered whether the trouble was a sequel of influenza or a consequence of oral sepsis. removal to a sanatorium was advised, and accordingly Mr. P. was sent to the Cotswold Sanatorium, under the care of Dr. Kincaid, to whom we are indebted for the further history of the case. It was on August 13, during his sojourn there, that the important further fact in the medical history was elicited, namely, that about two years previously he had had syphilis, for which he had been continuously under treatment for twelve months.

On admission to the sanatorium the mouth was found to be foul, and as it had been suggested as possible that the pulmonary infection was secondary to oral sepsis, and as on further examination no tubercle bacilli were found in the sputum, a dental surgeon was called in and the mouth cleared. The temperature and other symptoms, however, remained (Fig. 37). On or about September 10 headache became a marked symptom, and on September 14 Dr. Kincaid observed a definite swelling over the right frontal and parietal bones, having the characters of a syphilitic node. The temperature, as seen by the chart, continued to rise daily to 100° or 102°. Iodide of potassium, which had been commenced on first eliciting the syphilitic history, on August 13, but could only be tolerated for four days, was steadily resumed on September 14. The physical signs and symptoms now began rapidly to clear up. Mr. P.'s general health improved, his temperature became normal, and continued so during the remaining eight weeks of his stay at the sanatorium, with the exception of the two or three occasions on which mercurial injections were used, which gave rise to so much local irritation that they were discontinued. He left the sanatorium in the middle of December, 1909, having gained 3 stone in weight. There was only slight dulness remaining at the apex of the lower lobe of the right lung, and the cranial swellings had completely subsided.

Dr. Bulger reported in October, 1910, that since his return home he had continued well, and had resumed his work; and again in 1916 and at the end of August, 1918, that he was in good health.

The clinical interest of this case is obvious. There was some difference of opinion as to whether it was one of a chronic septic condition, in which the lung became involved, or of tuberculosis. The specific history, carefully concealed from his family attendant, was, at the time that he was first seen, revealed by no external sign. Hence the hectic features of the case, the cough, with on one occasion

blood-stained sputum, and the depressed opsonic index of resistance with regard to tubercle, were strongly in favour of the tuberculous view, which the absence of tubercle bacilli from the sputum on several examinations did not wholly refute. Nor did the pyrexial symptoms

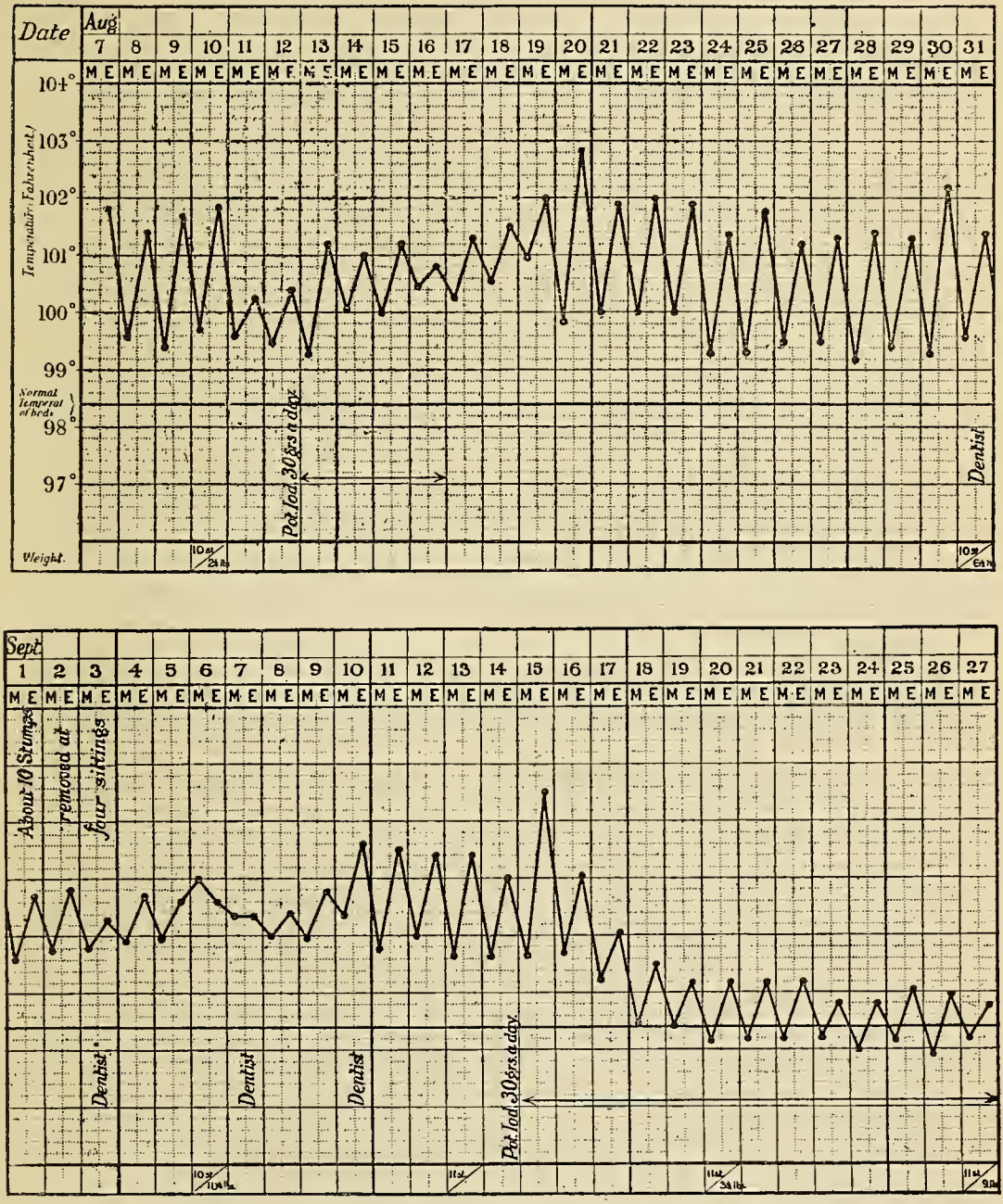


FIG. 37.—TEMPERATURE RECORD OF THE CASE OF SYPHILITIC DISEASE OF THE LUNG DESCRIBED IN THE TEXT.

promptly yield when, under the best hygienic circumstances, it was possible to clear the mouth, until the manifestation of definite external syphilitic phenomena imperatively indicated the use of iodide of potassium.

Treatment.—In the treatment of pulmonary syphilis it is well to commence with a course of salvarsan, provided that the heart and kidneys, and especially the liver, appear to be healthy. Neo-salvarsan is perhaps the best preparation to use, but if it cannot be obtained, galyl or neo-arsenobillon should be substituted. In the adult 0·6 to 0·9 gramme of neo-salvarsan may be injected intravenously with full antiseptic precautions once a week until six injections have been given. During the course mercury and iodide should also be prescribed. If the administration of mercury by the mouth causes bowel irritation, 5 minims of sterilised mercury cream (containing 1 grain of metallic mercury in 5 minims) may be injected once a week into the muscles of the buttock, three or four days after each of the salvarsan injections; or the drug may be administered by inunction. After the above course of injections the salvarsan and mercury should be stopped, but the iodide continued. After two months, if the Wassermann reaction is still positive, another course of mercury and salvarsan should be prescribed, and the iodide afterwards continued with intermitting courses of mercury, the Wassermann reaction being tested from time to time. In some cases a few seasonal courses at Aix-la-Chapelle may be recommended, and in all the general health of the patient must be well looked to and maintained.

Pulmonary Tuberculosis in Syphilitic Subjects.

More common than true pulmonary syphilis are those really hybrid examples of tubercle engrafted upon a more or less strongly marked syphilitic cachexia, in which the phenomena of syphilis are manifested with, and in a measure modify and control, those of phthisis. Cases of this kind present certain peculiarities. They are usually of the caseous pneumonic type, being one-sided, with rapidly advancing consolidation and softening, although arrest and fibroid changes may ultimately take place. Hæmoptysis is an early and an urgent symptom in a large proportion of cases, the hæmorrhage being severe and recurrent. The evidences of lardaceous change in other organs present themselves at a relatively early period, and with greater proportional frequency than in the other forms of phthisis. The patient may present other marks of syphilis, such as cicatrices upon the tongue or throat, nodes, cutaneous scars, or pigmentation. His build and conformation of chest

are often not those of ordinary phthisis, and the complexion has the earthy, lustreless pallor of the syphilitic, rather than the hectic aspect of the phthisical cachexia. In other cases the patient presents the aspect typical of advanced consumption, although the pulmonary lesions discoverable are but slight; in such instances excessive activity in anti-syphilitic treatment may have prostrated a patient of tuberculous constitution.

The treatment requires a judicious combination of anti-syphilitic with hygienic measures, which may prove successful in arresting the disease.

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[2] See—

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(2) "A Case of Infantile Interstitial Pneumonia," by A. W. Sikes, M.D., *Transactions of the Obstetrical Society of London*, 1906, vol. xlvii., p. 74.

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CHAPTER XXVI

STREPTOTRICHOSIS (ACTINOMYCOSIS) OF THE LUNG AND PLEURA

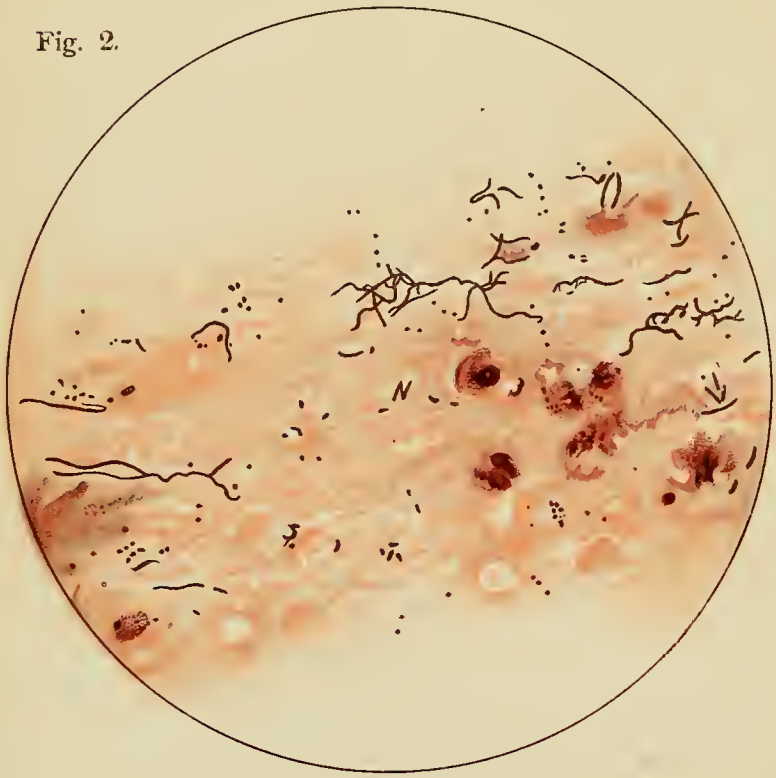
History of the Disease.—Streptotrichosis or actinomycosis, as it is still termed by many writers, whether attacking the lung or other organs, is no new disease; but until comparatively recent years cases of it would seem to have been mistaken for such varying maladies as cancer, osteosarcoma or tubercle.

The parasite was apparently first observed and figured by von Langenbeck of Kiel in 1845, in a case of caries of the spine, though the observations were not made known until many years later by Dr. James Israel.[1] In about 1855 it was independently observed and figured by the late Sir Thomas Smith,[2] though here again the drawings were not published until comparatively recently. Lebert,[3] in 1857, was the first actually to publish a description and illustration of the parasite, the organism in this instance being obtained from a case of pulmonary disease supposed to be cancerous, which terminated in abscess of the chest-wall. It was not, however, until 1878 that Israel[1] first definitely drew attention to the disease in the human subject. In the following year Ponfick,[4] showed the parasite to be closely allied—he believed identical—with the organism described by Bollinger,[5] in 1877, as the cause of the disease in cattle. From its radiate appearance, Hartz, a colleague of Bollinger in the Chair of Botany, had given to this latter organism the name "actinomyces," or "ray fungus."[6]

The Parasite.—Recent research has shown that the disease is produced not by a single parasite, as was once supposed, but by several organisms which are members of the streptothrix group. This group belongs to the Hyphomycetes, or lower mould fungi, and to the genus Discomyces, and is characterised by the following features, with which the writings of Mr. Alexander Foulerton[7] have made us familiar. The

Fig. 1.

Fig. 2.



Streptothrix Disease (Actinomycosis).

STREPTOTHRIX DISEASE (ACTINOMYCOSIS)

FIG. 1.—Specimen showing a small streptotrichial mass composed of a network of finely-branched mycelium; from the pus of an abscess in the chest wall connected with an empyema. (× 1010.)

FIG. 2.—Sputum from a case of streptotrichosis of the lung, showing the presence of scattered branching threads of a variety of Streptothrix. (× 1010.)

(Drawings by Mr. S. A. Sewell from preparations by Mr. Alexander G. R. Foulerton, F.R.C.S., stained by Gram's method and counterstained with eosin.)

PLATE XXIII

organisms consist essentially of filaments, which show true branching (Plate XXIII.), and are thus sharply distinguished from the Schizomycetes, or fission fungi, as represented by ordinary bacteria. The filaments in their growth tend to cohere, and to form little masses presenting a felted meshwork of finely branched mycelium (Plate XXIII., Fig. 1). They stain well by Gram's method, and in their early stages take the stain homogeneously throughout their length. Later the protoplasm within the sheath, which bounds the filament, tends to segment, and to present the appearance of longer or shorter rods, some of them still showing lateral branching. In the terminal filaments this segmentation is sometimes very marked, and rows of small round spores, still staining by Gram's method, are produced, the appearance closely resembling that of a chain of streptococci. Finally the sheath breaks down, and the spores and rod segments become free.

From time to time, when growing in the tissues, the mycelial mass may be seen to be surrounded by a radiating zone of club-shaped bodies, which do not stain by Gram's method, but may be counterstained by such dyes as orange-rubin or picric acid (Plate XXIV.). These "clubs" are probably not organs of fructification, as originally believed, but degenerate forms, produced when the soil in which the parasite grows is not especially favourable. In man their presence is very variable; in cattle they are more common.

In the discharges from actinomycotic lesions the organisms are often visible to the naked eye as characteristic scattered granules of greenish-yellow colour and of the size of small pinheads. They may be detected by tilting the pus along the side of the test-tube, or by spreading the sputum over the surface of a glass dish; but if not specially searched for they will almost certainly be overlooked. The granules are often very numerous, but their number tends to vary a good deal in the same case at different times. Under the microscope each little grain will be found to consist of a meshwork of the finely branched Gram-staining mycelium already described (Plate XXIII., Fig. 1), with possibly a zone of clubs surrounding it (Fig. 41, p. 402), but this is not common in man.

In a certain proportion of cases, however, the organisms are more disseminated, and do not form in the sputum the characteristic granular masses which are visible to the

naked eye. In such cases they may be most easily detected by staining by Gram's method and counterstaining with eosin. The branched mycelial threads of the variety of streptothrix present are in this way stained blue, and stand out well against the pink background (Plate XXIII., Fig. 2). An attempt should then be made by cultivation and animal experiment to complete the identification of the parasite, though the task may be by no means easy, since certain varieties of streptothrix grow only with great difficulty on artificial media. We may add that some of the strains prove acid-fast, staining like the tubercle bacillus.

Distribution.—Members of the streptothrix group have been found as saprophytes in water, in air, and especially in soil, and they have also been frequently met with in association with certain grasses and plants. In animals they have been found to be the cause of disease in many of the mammalia, notably in cattle; and in man several species have been isolated. Mr. Foulerton[7b] succeeded in differentiating ten varieties of streptothrix organisms in a series of fifty-three cases investigated by him. If the tubercle bacillus be classified as a streptothrix, in support of which a good deal of evidence may be advanced (p. 422), the pathogenic interest of the group becomes enormously increased.

Pathological Anatomy.—As the result of the introduction and growth of one or other species of streptothrix, an inflammatory reaction of the tissues is induced. Around the fungus, and about any centres whither its offshoots may be conveyed, the structure of the affected organ becomes densely infiltrated with granulation tissue, in which epithelioid cells, and sometimes giant cells, may be seen. This is at first freely permeated with new vessels, but caseation, degenerative softening, or suppuration speedily ensue, coupled, however, with some tendency to repair as shown by the formation of fibrous tissue. The disease in these respects closely resembles tubercle, but it differs, as we shall see, in the remarkable disposition shown by it to transgress the bounds of the organ affected, and to involve adjacent parts. The malady is further spread by the transference of the elements of the fungus through the blood-stream to distant localities, where secondary foci of the disease are generated.

Clinical Features.—Streptotrichosis *primarily* affecting the lung or pleura—and it is sometimes difficult to say in which it

has really commenced—is a comparatively rare disease, though no doubt cases have been overlooked. More usually the lung is affected *secondarily*, by direct extension from the liver or other adjacent organs.

The following case, in which the disease probably originated in the pleura and spread thence to the lung, will serve conveniently to bring together the more important clinical features. It is interesting to note that this was apparently the first case in this country in which the disease was recognised during life.*

Case I.—George W. H., a thin, well-featured boy, aged nine, with bright eyes, dark hair, long eyelashes, and somewhat hectic appearance, was admitted into the Brompton Hospital on October 8, 1888, under the care of Dr. Douglas Powell, complaining of pain of a pleuritic character below the right nipple.

The boy was of healthy parentage, his father, mother, and three brothers and sisters being all well, but there were cases of tuberculosis amongst the uncles and aunts on both sides. His father worked at a dairy as milker and carrier. The boy himself had enjoyed good health up to four months before admission. His parents attributed the commencement of his illness to a blow behind the ear received at school early in June, which kept him at home for a week, with headache and constipation. He returned to school, however, until the holidays commenced, in July, when he was sent to Brighton for a month, but returned only slightly better in August. For the six weeks following his return from Brighton the boy accompanied his father two or three times a week to the cowshed, where there were about 150 cows, remaining there two or three hours at a time. The father knew of no illness† which had lately affected any of the cows and was sure that none had died during the past year. It was towards the end of August, six weeks before admission, that he complained of languor, disinclination to work or play, and some pain and tenderness of the right side of the chest when touched or washed. A little later he developed slight headache, with some hacking cough, but with no expectoration or hæmoptysis. Throughout his illness he had gradually lost flesh, and during the last month he had had night-sweatings and his breathing had become shorter.

On admission the right side of the chest was obviously larger (1 inch by measurement) than the left, and restricted in movement. About the mammary region the intercostal spaces were obliterated

* The case is recorded in the *Transactions of the Royal Medical and Chirurgical Society*.[8]

† Professor Crookshank and Mr. Taylor visited the dairy on November 17, and found the cows looking healthy, and were assured by the owner and by two of the milkers whom as well as the boy's father they questioned, that there had been no cases of "wens" or "clyers" or other manifestations of streptothrix disease on the farm.

by a smooth and rather hard swelling of the tissues, having its centre in the fifth space in the nipple line (Fig. 38, A). The swelling was exceedingly tender and slightly œdematous, but at this time no distinct fluctuation could be felt, nor was there any blush of redness on the surface. On sitting the boy up, the head was observed to be inclined to the affected side, and the scapular angle correspondingly tilted inwards, the spine being slightly curved in the dorso-cervical region, with concavity to the right. In the posterior axillary line a second larger swelling was observed, corresponding with the seventh, eighth, and ninth ribs (Fig. 39, B). This swelling was also tender, brawny to the touch, and suggestive of the early stage of a

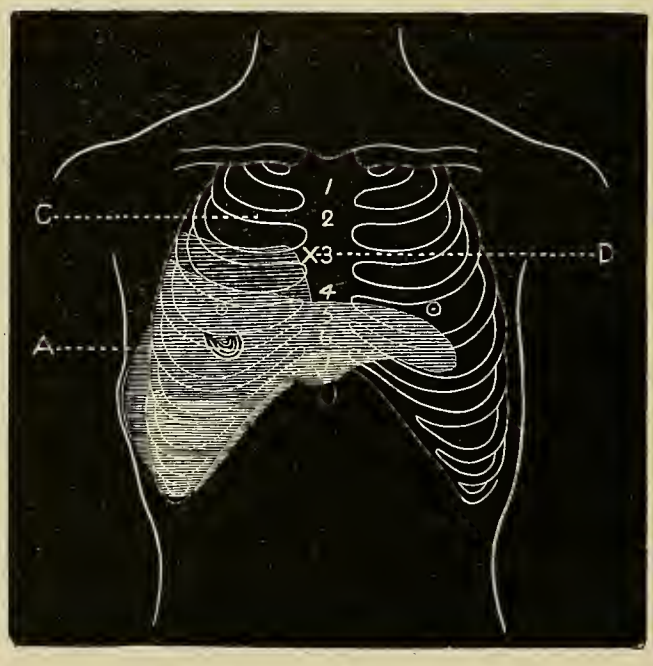


Fig. 38.—Diagram showing the Physical Signs in the Case of Streptotrichosis of Lung and Pleura described in the Text.

A, Pointing of prominent and softened tissues below the nipple (the lateral bulging and shading extending beyond the confines of thorax proper shows infiltration of the chest wall); C, skodaic resonance; D, systolic murmur.

pointing empyema. The heart's apex-beat was half an inch to the left of the nipple line.

The percussion note on the right side was resonant, of somewhat skodaic quality, to the second place in front (Fig. 38, C); below that point, dull, the dulness being complete at the fourth cartilage. Posteriorly there was dulness from the lower third of the scapula to the base. It was observed, however, that the upper line of dulness was not horizontal, but slanted downwards towards the spine, so as to be at a lower level there than in the mid-scapular and axillary lines. In the subclavicular and superior scapular regions the respiratory murmur was weak but vesicular, but over the dull area generally the

breath-sounds were absent and the vocal fremitus was lost. On the left side the resonance and respiration were good.

A short systolic murmur was audible at the right third cartilage (Fig. 38, D); otherwise the heart-sounds were natural. The liver dulness blended with that of the thorax, and did not extend below the cartilages, nor could the margin of the liver be felt. The spleen was normal.

From the hectic temperature and symptoms and the physical signs above related the case was regarded as one of empyema. Two punctures with a fine needle, however, in the fourth and fifth spaces in the anterior axillary line produced only a little blood.

October 15.—Whilst under chloroform for more complete explora-

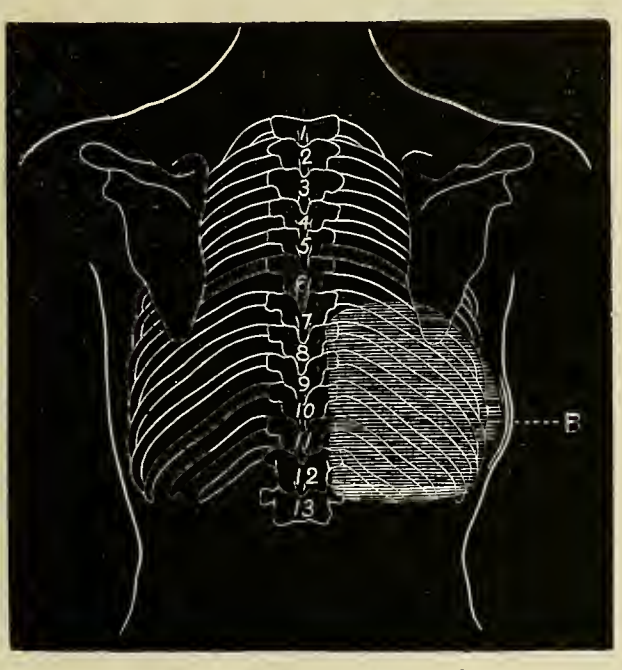


FIG. 39.—DIAGRAM SHOWING THE PHYSICAL SIGNS IN THE CASE OF STREPTOTRICHOSIS OF LUNG AND PLEURA DESCRIBED IN THE TEXT: POSTERIOR VIEW.

At B infiltration and bulging of chest wall are shown.

tion, but before any puncture was made, the patient expectorated about half a dozen sputa of bright blood. A fine trocar was introduced by Mr. (Sir Rickman) Godlee at the centre of the posterior bulging, and some drops of blood came out through the cannula, which on examination contained only fibrin and blood-cells. The temperature from this time onwards is shown on the chart (p. 400). The patient as a rule slept and took food fairly well, and was very placid and cheerful, being free from pain except when touched or on coughing. The cough was troublesome, but without expectoration.

By the end of the month he had notably lost flesh since admission, and the appearance of hectic was more marked. The swellings had become more prominent and were distinctly elastic; and extending

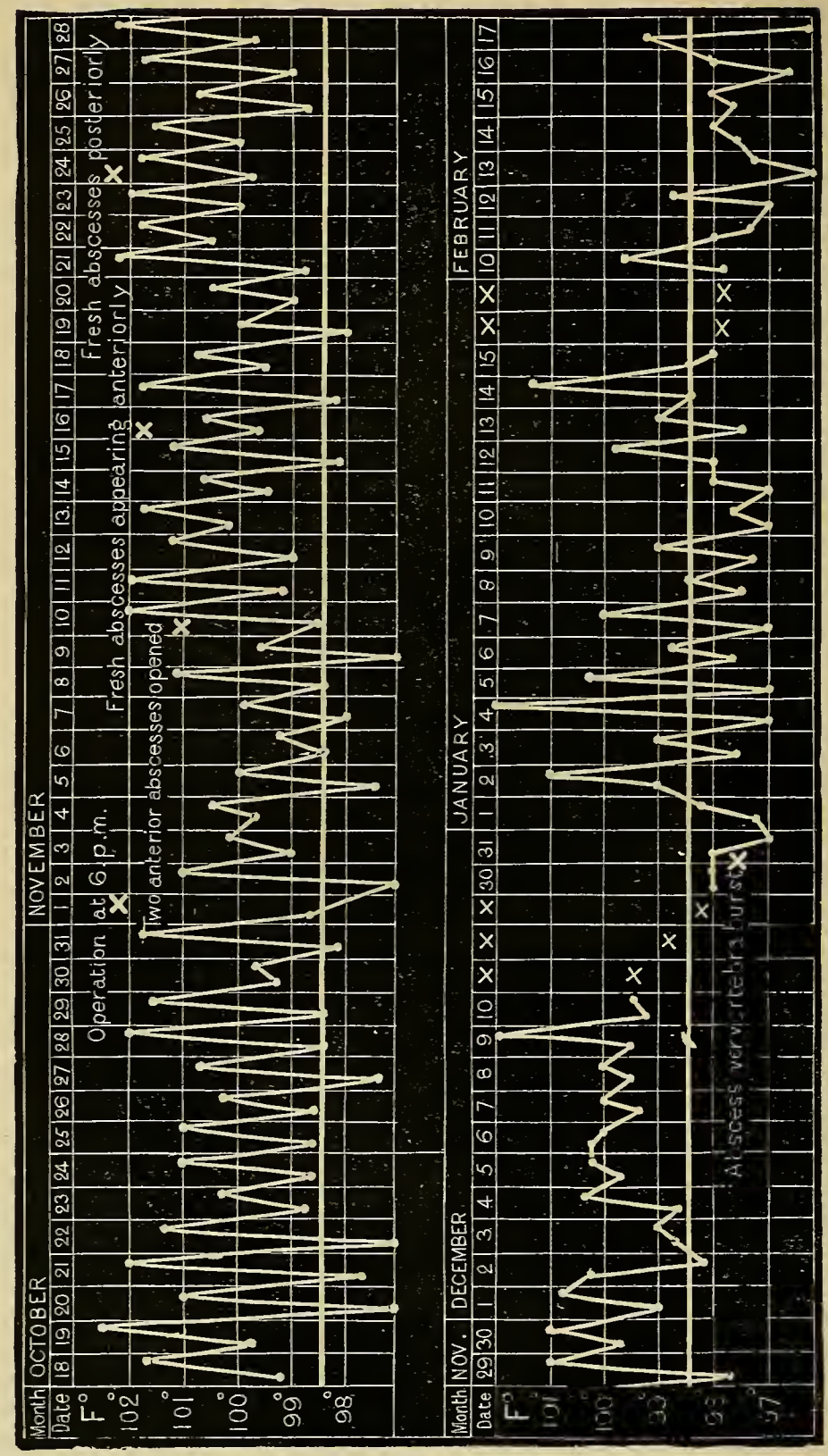


FIG. 40.—TEMPERATURE CHART TO ILLUSTRATE THE CASE OF STREPTOTRICHOSIS OF LUNG AND PLEURA DESCRIBED IN THE TEXT.

Fig. 1.

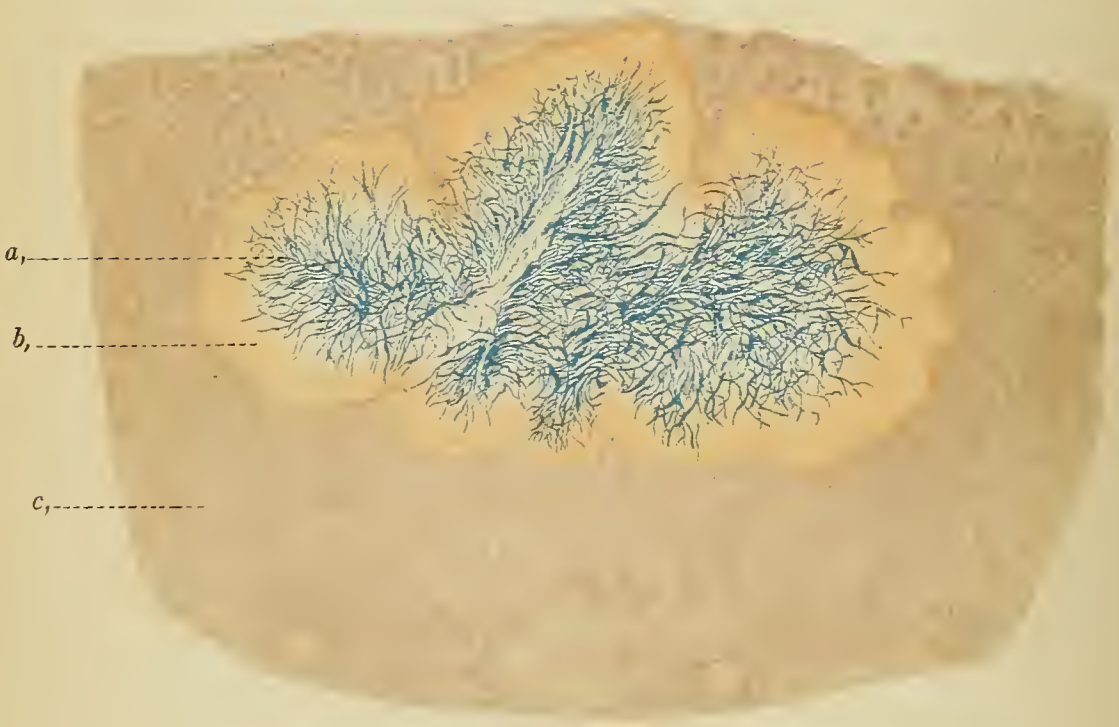


Fig. 2.

Streptothrix Disease (Actinomycosis).

STREPTOTHRIX DISEASE (ACTINOMYCOSIS) OF THE LUNG AND PLEURA

FIG. 1.—Specimen from pleura under low power. (1″ obj.)

FIG. 2.—The same more highly magnified. ($\frac{1}{4}$′ obj.)

a. Central network of branching mycelium.
b. Palisade of clubs.
c. Surrounding dense fibro-nucleated tissue, plentifully supplied with bloodvessels.

(From drawings by Dr. Wynter; stained by Weigert's modification of Gram's method and orange rubin.)

PLATE XXIV

downwards and slightly forwards from the original seat of the posterior swelling was a pyriform bulging, three inches in length, with the larger end depending, which yielded semi-fluctuation over its whole area, but no impulse on cough. There was some redness over the posterior swelling. The physical signs remained much the same. Two glands, slightly enlarged and movable, were to be felt in the right axilla, and on this side of the neck along the anterior margin of the sterno-mastoid some slightly enlarged glands were also perceived. On the opposite side the glands were not enlarged. The systolic murmur at D was distinct, short, and rough.

Except for slight labial herpes on admission the patient had no sores on the face or mouth. The milk molars were much decayed, but the permanent teeth sound.

At this time it was thought that there was malignant growth in the thorax, but the idea of there being pus in the pleura was not wholly abandoned.

On November 1 the upper and posterior of the two swellings was thoroughly explored with the aspirator by Mr. Godlee, but only some soft caseous material was found in the cannula. An incision was made into the swelling, and a large collection of similar material with some fresh blood was found between the ribs and the skin. An inch of the sixth rib above the incision was removed, the pleura incised, and the opening having been dilated by dressing forceps, the finger was passed in, and entered a soft collection of the same material as had been found outside. No limitation of the mass could be felt; some partially clotted brain-like material mixed with blood escaped, but there was no actual flow of fluid matter. The bleeding was considerable. Strips of lint soaked in sublimate lotion—1 in 2,000—were inserted to plug the wound, and a dressing of carbolic gauze was applied. A little blood was expectorated towards the end of the operation.

November 2.—The patient passed a fairly good night; there had been no hæmorrhage, and he was comfortable. Portions of the crude material removed on the previous day were examined by Mr. Taylor. Fresh sections taken from the brain-like material were made the same evening and stained with alum-carmine. The structure generally was found to be of the nature of granulation tissue, finely fibrillated, giving at parts a sponge-like appearance and containing many vessels. There were also observed several deeply stained bodies having the recognisable characters of actinomyces. At the earliest opportunity specimens hardened in alcohol were stained by Gram's method, and the fungus was demonstrated in its entirety, exhibiting both threads and clubs, the latter, however, not taking the stain (Plate XXIV.).

A purulent discharge escaped from the wound, which was treated with iodoform and antiseptic lotions.

November 10.—It was found that the lower swelling communicated with the upper, and it subsequently burst externally through a small opening leading to a sinus which communicated with the upper

wound. On pressing on the surrounding tissues thick blood-stained pus freely escaped, and was seen to contain innumerable minute white granules resembling the bodies found in bovine actinomycosis (Fig. 41). Professor Crookshank, who was kind enough to assist us in the investigation of the case, collected the pus, and inoculated a number of tubes of various nutrient media. The surface of the wound was also covered with yellowish-white granules, glistening and not readily removed. When scraped off, the material was found to contain iodoform from the dressings and abundant ray fungi.

The boy remained free from pain, cheerful, and placid, but his emaciation increased, and the pulse quickened to 130, although the respirations were quiet. There was no enlargement of the liver. The upper level of dulness reached the second rib, and the level did not alter with position. Some rather fine crackles were heard over the second and third ribs, with very weak breath-sound. Posteriorly the dulness

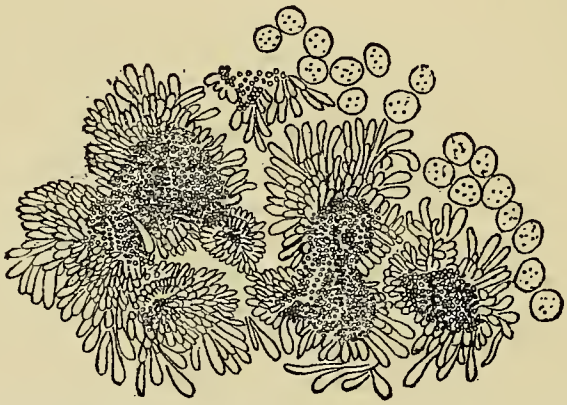


FIG. 41.—STREPTOTRICHOSIS OF LUNG AND PLEURA. COVER-GLASS PREPARATION FROM THE PUS, UNSTAINED, MOUNTED IN GLYCERINE, × 350 (ABOUT), SHOWING THE RADIATING ARRANGEMENT OF THE CLUBS AROUND THE MORE CONFUSED MYCELIAL CENTRES.

reached to the middle of the scapula, above which level the breath-sounds, although weak, were fairly good.

On injecting carbolic lotion into the wound the patient coughed and expectorated a frothy muco-purulent sputum.

November 16.—Under chloroform two anterior abscesses were opened, and the opening below the original incision was enlarged. Thick yellowish pus with characteristic granules escaped freely; and there was considerable hæmorrhage, causing some degree of collapse. A little of the more solid material was removed by scraping. Professor Crookshank was present with sterilised tubes, and took a considerable quantity of pus and thick material for inoculation into animals. The whole of the right thorax was more or less puffy, and along the ribs, both above and below the front incision, were several points of fluctuation.

The wounds all had a languid appearance, edges purplish, granulations pale and large; a thickish pus adhered to them in places, on

which, and on bare granulations, were seen minute dirty yellowish granules, varying much in size, one of them as much as a millimetre across. These were readily shown by microscopic examination to be portions of fungus. As yet no growth had protruded from the wounds. Many granules were visible on the dressings, and some doubtful spots were seen in the expectoration, which was still scanty.

November 20.—On this date the fungus was found in the sputum for the first time. No tubercle bacilli had at any time been detected. The urine, of specific gravity 1028, acid, gave a decided indican reaction, and rapidly manifested decomposition, but contained neither albumin nor sugar.

With some variations from day to day, the boy gradually lost ground, becoming more emaciated and pale.

December 18.—Several swellings had appeared upon the back, all of which fluctuated, but as they were not painful, they were not incised. The wounds from the former incisions presented a thick, yellowish-white, slightly glistening surface, and similar masses were fungating through other places in which abscesses had spontaneously given way.

Through January there was some general improvement in the patient, and the activity of the growths seemed somewhat checked, but after several partial rallyings and relapses, he sank exhausted on February 26, 1889.

Abstract from the Post-Mortem Report[9a]—*Thorax.*—A large mass of soft material was found to occupy the pleural cavity of the right side from about the level of the second rib downwards. It was very soft, pultaceous, and canary-coloured, much resembling coarse, badly-made mortar, and appeared to consist of the same material in varying stages of degeneration. Between the base of the lung and the liver there was a large amount of similar material, with which the diaphragm appeared to have become incorporated. In the upper part of the pleura there were some recent adhesions.

Lungs.—Right: the lower anterior part of the lower lobe and the middle lobe were converted into tough fibrous material, which had an irregular interlobular distribution, the bands of which enclosed small pinkish areas like altered lung tissue. Here and there in the midst of the fibrous growth there were irregular, canary-coloured masses like that above described. The upper lobe was plentifully studded with grey nodules, the size of a hemp-seed, some slightly pigmented, and exactly resembling tuberculous nodules. Left lung: aërated throughout, but containing scattered nodules, of the size of a pea to a small Spanish nut, which, on section, had the characteristic canary-coloured appearance.

Abdomen.—Between the liver and diaphragm there was a collection of pultaceous matter similar to that described above. The liver itself appeared healthy, but on section about one inch below the upper surface an abscess the size of a small orange was discovered, having a fibrous tissue wall about half an inch thick. The

contents consisted of tenacious yellow pus, which had a trabeculated appearance.

The right lateral sides of the dorsal vertebræ were covered over by the growth and superficially eroded. Some of the ribs, between which the growth had passed to reach the outside of the chest, were also eroded.

Nothing worthy of note was found in any other of the organs.

Remarks.—The ætiological history of this case does not throw any definite light upon the exact mode in which the malady originated. The occupation, however, of the father, and the fact that the lad apparently often accompanied him in his visits to the cowsheds, rendered possible an infection from a vegetable source, which is often observed in these cases.

Even after post-mortem inspection it was not easy to decide exactly where the disease originated, although it is clear that it flourished chiefly in the lower two-thirds of the pleural cavity, infiltrating the chest wall and fungating through the cutaneous surface, denuding the adjacent vertebræ, and commencing to invade both the diaphragm and pericardium.

The lung was collapsed backwards and upwards, toughened and fibroid, containing also many centres of the parasitic disease, but there was not sufficient evidence of any considerable primary fungus disease of the lung with subsequent cicatrisation. On the contrary, the post-mortem evidence rather favoured the view of the compressed lung having become the seat of a secondary actinomycosis; the left lung, otherwise healthy, also presented centres of a similar kind in an early stage.

On the whole, it would seem probable that the organism, having gained entry through the respiratory tract, had been conveyed through the lymphatics to the pleural surface, and there germinated and flourished in inflammatory materials derived from the pleural layers and their subjacent tissues, the bulk of the growth being in the thoracic wall.

The clinical features of the case, both as regards the symptoms and signs, were in the first instance those of empyema. On puncture, however, no pus could be obtained, and on further exploration the evacuation of semi-clotted material, resembling a mixture of fluidified brain-substance and blood, quite unlike anything which had in our experience been removed from cases either of intrathoracic growth or empyema, seemed to justify the removal of a portion of the rib for more thorough exploration of the pleura. The case now presented the appearance of some degenerating growth, but certain signs which have been above related—and especially the somewhat slanting and shifting upper line of dulness and the skodaic resonance below the clavicle—led us still to retain the hope that there might be some fluid collected behind a thick, caseous layer in the pleura, or *possibly* connected with some degenerated growth in the pleura, such as we had once before met with in a case of dermoid tumour of the lung. At

the end of the exploration, however, we returned to the belief that we were dealing with an anomalous degenerated and softened malignant growth of extreme vascularity. It was only on careful examination by Mr. Taylor of the material removed that a diagnosis was arrived at.

The following case also possesses features of sufficient interest to be here recorded:

Case II.—Mrs. C., aged forty-five, five years married, no family or pregnancies, was first seen by Sir R. Douglas Powell, in consultation with her friend and family attendant, Dr. Waithman, towards the end of April, 1903, on account of a severe pain of a neuralgic character in the right shoulder and radiating therefrom. Mrs. C. had suffered from epileptic attacks, for which she had taken bromide, the last fit having been about February, 1903. She had also had some slight uterine troubles; otherwise she had never been ill until an attack of supposed whooping-cough in 1901, which was followed by some pleurisy, with cough and night-sweatings, which gave rise to the suspicion of commencing tuberculosis. The cough was not attended by any expectoration. She spent the winter of that year at Nice.

The winter of 1902 was passed in Egypt, going by dahabieh up to Luxor, and during that time Mrs. C. suffered greatly from the pain in the shoulder, which was regarded as a rheumatic neuritis and treated by salicylates, and by increasing doses of morphia given subcutaneously. During her stay at Luxor it was stated by Dr. Appleby, under whose care she then was, that her evening temperature was never below 100°, sometimes 102°, and that she had heavy night-sweatings. The above treatment was continued on her journey home, via Naples, during which she underwent great suffering. The history of the pleuritic attack and the irregular pyrexial and hectic manifestations were in support of the belief that her illness was tuberculous.

When first seen by Sir Douglas Powell, in April, 1903, on her return from Egypt, some impairment of the percussion note was observed below and over the right clavicle, and the breath-sounds were noted to be harsh and wavy, and a few râles were heard. On sitting the patient up and passing the hand over the supraspinous region on this side attention was at once attracted to a fulness, and on deepening the pressure, considerable tenderness was elicited; a sense of elastic tension was appreciable, but no distinct fluctuation. The fulness was also apparent on inspection, compared with the other side. An abscess connected either with caries of the upper dorsal vertebræ, or growth, or actinomycosis, was diagnosed, and as the pain was very severe, rendering nights sleepless, except after strong doses of morphia, and days wellnigh intolerable, the aid of Mr. (Sir Rickman) Godlee was sought. He made an incision between the superior angle of the scapula and the spinal column, passing deeply through the

several layers of muscles, until finally some purulent matter welled forth, which on examination revealed the characteristic grains of actinomycosis. The pleura was reached, and found adherent, there being no pleural collection.

There was no obvious connection between the abscess and the lung, but the fact that the lung was involved in the disease was inferred from the physical signs. No expectoration was at this time obtainable for examination, and only later were the characteristic granules discovered in the sputum. The pain was at once so far relieved that no further morphia was necessary. The abscess continued, however, for a long time to discharge through a fistulous tract. After recovery from the first operation the patient was put upon iodide of potassium in doses of 30 grains a day, afterwards increased to 60 grains.

Mrs. C. went to Scarborough in July, 1903, and subsequently to her home in Yorkshire, living out of doors. She gained weight and improved generally, but towards the end of September Dr. Fisher, of Skipton, reported: "For the past ten days Mrs. C. has not been quite so well, suffering more pain in her chest and tenderness all over the side. She cannot localise it. Five or six days ago pleuritic friction could be distinctly heard just behind the shoulder-joint; to-day it is almost gone, and she has less pain and discomfort. Her cough is still troublesome at nights, but there is no expectoration. Since September 9th her temperature, normal in the morning, has ranged from 99° to 100° in the evening. Between August 1 and September 19 she has gained in weight from 11 stone 3 pounds 2 ounces to 11 stone 11 pounds 7 ounces. She takes from three to four pints of milk each day, also her full doses of the iodide. Last week she had two or three profuse perspirations at night. The wound in the back is still open and discharges a little. She is out in the garden in her chair each fine day." Dr. Waithman saw her about this time and agreed with the above note, and after consultation with Dr. Fisher and Sir R. Douglas Powell, it was decided for her to go to Torquay for the winter. When passing through town in October, 1903, a second incision, opposite the infraspinous portion of the scapula, was found necessary to relieve some pent-up pus, and a third operation was required in February, 1905, eighteen months later, before the wounds finally healed.

In the later periods of surgical treatment, the details of which were carried out with Mr. Godlee's usual care and resourcefulness, and require no special mention, peroxide of hydrogen lotion was syringed into the wounds, and subsequently iodoform emulsion, but on no occasion did any such lotion find entry to the lung. The purulent discharges were repeatedly examined, and always showed the characteristic granules and microscopic appearances of streptotrichosis, including the club-shaped bodies.

The sputum, which at first could not be obtained, was found on October 23, 1903, to present the following characters: it was composed of viscid mucoid material, with streaks of muco-pus; and

contained also some small opaque grains of a yellowish colour, the largest about the size of a small pin's head. Microscopically examined the granules were found to consist of a felt-work of mycelium, which retained the Gram stain, and from which projected some more refractive bodies (presumably clubs). No tubercle bacilli were present.

The iodide of potassium was, with occasional breaks, continued for the two years of treatment. Hygienically Mrs. C. was treated on fresh-air lines, mainly between Yorkshire and the South of England. The wounds finally healed in 1906. Her lung signs, which never gave serious trouble, cleared up, and her weight increased from 10 stone in June, 1903, to between 11 and 12 stone, at which it has since remained. Except for occasional epileptic seizures she has since continued in good health, and Dr. Waithman informs us that she is well at the present time, June, 1919.

Mrs. C. was not a horsewoman, and beyond visiting the home farm, there was no special connection with straw or herbage.

Chief Features of the Disease.—Since the occurrence of the first of the two cases above recorded, not a few instances of primary streptothrix disease (actinomycosis) of the lung or pleura have been described, and the malady is probably more common than is usually supposed. Some years ago Dr. Samuel West[10] collected thirty from the literature, three more have occurred at the Brompton Hospital,[11a] and during the years 1900-1912 Mr. Foulerton[7a] himself observed fourteen at the Middlesex Hospital.

The disease may occur at any age, and, as with streptotrichosis of other organs, is more often met with in the male than in the female. Both lungs may be affected, but the left is more commonly the seat of the disease than the right, and usually, though by no means always, the lower part of the lung is first attacked. The infection may, however, commence at the apex, as in the second case which we have described, and the disease then closely simulates pulmonary tuberculosis. This occurred also in one of the Brompton cases,[11b] in which the physical signs at the right apex were strongly suggestive of early tuberculous disease, and were believed to be of that nature until streptothrix granules were found in a secondary abscess, and were then at once looked for and discovered in the sputum. At the autopsy in this case the appearance of the lung, showing fibrosis, most marked at its upper portion, with areas of softening and scattered patches of broncho-pneumonia,[9b] closely resembled that seen in cases of pulmonary tuberculosis. In other instances the areas of individual disease

are larger, and the involvement of adjacent lung less marked than is commonly seen in tuberculosis[12] (Plate XXV.).

If the pleura be affected an empyema generally results, in the pus from which the parasite is found. The occurrence of pultaceous material in the pleural cavity without suppuration, as in the case above related, is not common.

In its intimate pathology, as we have seen, streptotrichosis is closely allied to tubercle, yet in one important feature it shows a striking difference, namely, its disposition to invade beyond the borders of the organ first attacked, and to pass "across country," so to speak, through adjacent tissues to other parts and other organs. In this way the lung is not infrequently invaded through the diaphragm from the liver, and in this way, too, arises that infiltration of the chest wall and the formation of those secondary abscesses which are so often seen in streptotrichosis of the lung and pleura. Sir Rickman Godlee[11a] calls attention to the small amount of pus generally contained in the abscesses, and their "indefinite spongy walls, easily breaking down before the finger in all directions, and bleeding very freely," features which he regards as characteristic.

There is one other feature of the complaint to which we must refer, namely, the tendency for the organisms to pass into the blood-stream, and to set up infective abscesses in distant parts, such as the brain, liver, and kidney. This "pyæmic form" of the disease, as it is termed, is not very uncommon, and the same disposition has been observed when other organs than the lung are primarily attacked.

Diagnosis.—Pulmonary streptotrichosis should at once be suspected if in a patient with symptoms of chest disease induration or suppuration of the chest wall makes its appearance. The finding of the parasite in the pus or sputum will complete the diagnosis.

It should be a rule also to bear the possibility of streptotrichosis in mind in all cases which suggest pulmonary tuberculosis, and especially if the signs be atypical or basal in character, but in which repeated examination of the sputum has failed to reveal the presence of tubercle bacilli. In all such cases careful search should be made for streptothrix organisms.

Treatment. — Pulmonary streptotrichosis in the human

PLATE XXV

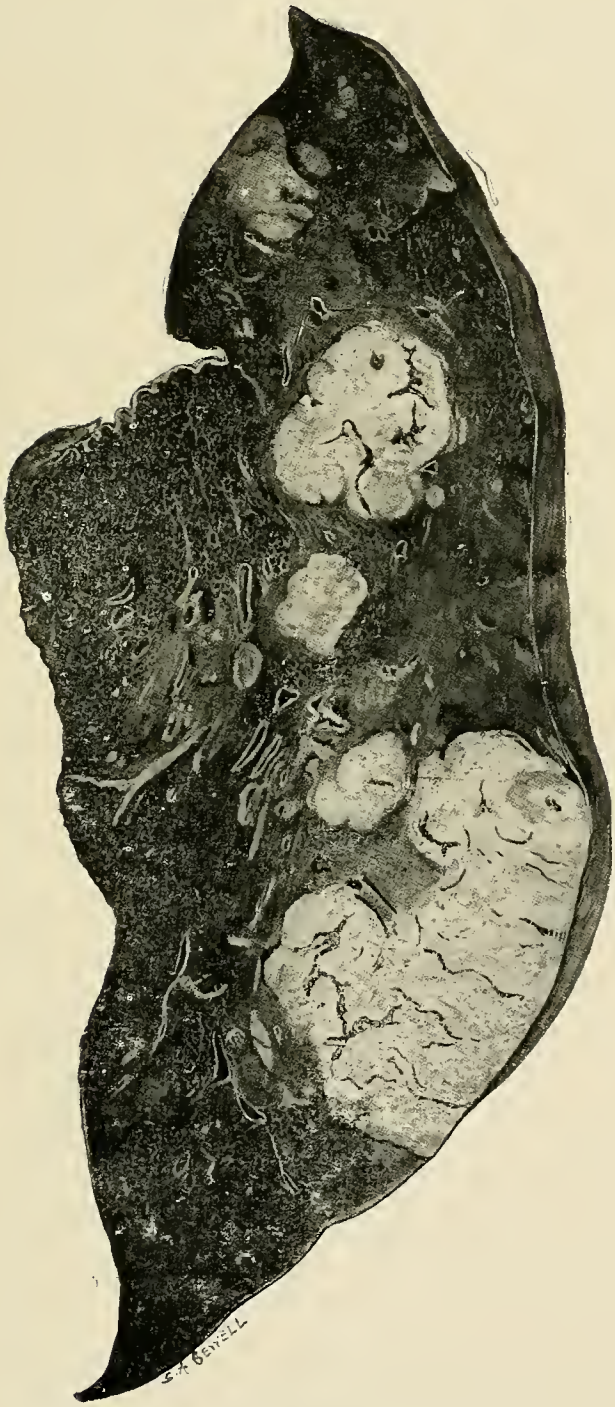
STREPTOTRICHOSIS OF LUNG

The drawing shows a section of the right lung. Near the base of the lower lobe a large white mass of irregular outline is seen, composed of caseating material honeycombed with suppurating foci. Similar smaller masses are seen scattered through the lobe; some were also found in the middle lobe, but none in the upper lobe or in the left lung. Microscopical examination of the purulent material from the lung revealed the presence of a Gram-staining and acid-fast streptothrix, which grew well on artificial media and was pathogenic to guinea-pigs and rabbits. It closely resembled the organism originally described by Eppinger.

From a porter aged sixty-seven, who died after an acute illness of five weeks' duration. The case is described by Dr. J. M. Bernstein in the *Proceedings of the Royal Society of Medicine*, vol. ii. (Pathological Section), p. 271.

(From the Museum of the Royal College of Surgeons.
Natural size.)

PLATE XXV



STREPTOTRICHOSIS OF LUNG.

To face p. 408.

subject, if untreated, tends to pass rapidly from bad to worse. It is important, therefore, at the earliest moment to commence treatment with considerable doses of iodide of potassium, the only drug which has so far been known to exert any influence upon the disease. The administration of the drug should be pressed, twenty to forty grains being given three times a day, and sometimes even larger doses may be prescribed with advantage.[13] In many cases associated suppuration calls for surgical treatment, and very favourable results have in some instances followed this combination of medicine and surgery. It cannot be said, however, that iodides are always successful, for the drug has in some cases seemed to produce no effect. This may possibly be due to the special variety of streptothrix concerned in the disease, but probably also to the malady not being recognised and boldly attacked at a sufficiently early stage. These measures may be supplemented by vaccine therapy. At first a stock vaccine should be employed, to be replaced later by an autogenous vaccine, if it be found possible to grow the streptothrix discovered in the patient. Cases of streptotrichosis have been successfully treated on these lines,[14] but we have known of others in which no apparent benefit has resulted. In many cases, all attempts at cultivation have failed, so that the preparation of an autogenous vaccine has been impossible.

The patient's diet should be nutritious and sustaining, port-wine or brandy being freely given when indicated, with such drugs as bark, quinine, or other tonics. Special symptoms must be treated as they arise.

REFERENCES.

[1] "Neue Beobachtungen auf dem Gebiete der Mykosen des Menschen," von Dr. James Israel, *Virchow's Archiv*, 1878, vol. lxxiv., pp. 15 and 50, Plate III., Fig. 9.

[2] "A Fading Record: Early Observations on the Ray Fungus by Mr. Thomas Smith, F.R.C.S.," by A. A. Kanthack, M.D., M.R.C.P., *St. Bartholomew's Hospital Journal*, 1896, p. 50.

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CHAPTER XXVII

SPOROTRICHOSIS

THIS disease was first described by Schenck[1] in 1898 in the case of a man aged thirty-six, attending the Johns Hopkins Hospital, Baltimore, and has since been chiefly studied in France by de Beurmann[2] and his pupils. It is produced by various closely allied filamentous spore-bearing fungi belonging, like the Streptothrix, to the Hyphomycetes or lower mould fungi, but to the genus Sporotrichum. The varieties usually met with are the Sporotrichum Schencki and the Sporotrichum Beurmanni, which are possibly identical, but others are described.

In the pus and tissues the parasites are found chiefly as short oblong forms resembling bacilli. When cultivated these yield a delicate septate branched mycelium which soon shows spore formation, the spores being arranged in clusters around the end of a filament, or less commonly along its sides. The organisms stain with the usual dyes and grow well at room temperature on Sabouraud's glucose-peptone gelatine, which should be used for their isolation.

The parasite is found widely disseminated in nature, and has been detected on lettuces, potatoes, grain, bark, and thorns, and is thus easily conveyed to man. Infection usually occurs in patients with lowered vitality, and is produced through an abrasion of the skin, but in the pulmonary cases inhalation may be the mode of entry as in the case of a woman who attributed her complaint to her dusty occupation in a coffee mill.

The characteristic lesion manifests itself as a small hard nodule or gumma in the subcutaneous tissue, which after some enlargement undergoes central softening and abscess-formation, or becomes converted into an ulcer. The ulcer is of chronic type, and suggests a syphilitic or tuberculous origin.

The lesions affect chiefly the limbs, and especially the hands and arms, but the disease may become disseminated and affect the face as well as the trunk.[2c] Sporotrichosis is not, however, confined to the subcutaneous tissues, and may attack the bones and joints, the muscles, the epididymis, the eye, the mucous membranes of the mouth and larynx, and occasionally the lung, in which case the symptoms usually suggest a chronic pulmonary tuberculosis.

The following case of the rare pulmonary variety of the disease, which recently occurred in Paris, has been recorded by Drs. Schulmann and Masson:[3]

A workman, aged thirty-six, developed in 1911 an eruption on the face, neck, and chest, soon spreading to the extremities. The eruption was of chronic type, suggesting syphilis, but the Wassermann test was negative. The gummata enlarged, and in pus obtained from one which had softened the sporothrix was found. The patient was treated with iodide of potassium with benefit, though recovery would appear to have been slower than is usually the case, but in January, 1913, it is stated that the lesions were all cicatrised with the exception of one on the leg, and in March, 1914, that, save for some conjunctivitis, recovery was complete.

The patient appears to have remained well until May, 1916, when, during service in a munition factory, he began to cough and expectorate, to lose weight and to become febrile. Pulmonary tuberculosis was diagnosed, but tubercle bacilli were never discovered in the sputum. Some improvement followed visits to Cannes and Mentone, and he was able for a short time to resume work, but in November, 1917, it became necessary to discharge him from military service, and his symptoms becoming aggravated he was in May, 1918, admitted to hospital at Bicêtre. He was then much emaciated and his appearance strongly suggested phthisis. Examination of the chest showed extensive loss of resonance over the right lung, especially marked over the centre of the chest, with moist sounds at each apex. X-rays revealed diffused shadowing over the right lung, with some mottling, and in the left lung, extending from the heart region almost to the diaphragm, the well-defined shadow of a somewhat deep-seated oval tumour was visible. No tubercle bacilli could be found in the sputum. The history of the former cutaneous lesions now raised the suspicion of pulmonary sporotrichosis, and the diagnosis was effected by lung puncture into the point of maximum dulness on the right side of the chest; the drops of blood-stained fluid thus removed were inoculated upon Sabouraud's medium and the Sporotrichum Beurmanni recovered, apparently in pure culture.

This case proves that pulmonary signs and symptoms resembling those of phthisis may be produced by the action of

the sporothrix, and renders it probable that other cases in which sporotrichosis has been diagnosed from the discovery of the parasite in the sputum may have been true examples of this disease. It must be remembered, however, that the sporothrix may sometimes occur as a saprophyte in the pharynx, and thence find its way into the sputum, so that its mere presence in the expectoration is not in itself conclusive evidence that the pulmonary disease is of this nature.

Diagnosis and Treatment.—The possibility of pulmonary sporotrichosis must be borne in mind, especially when, in addition to symptoms of chest disease, chronic cutaneous lesions, suggesting a tuberculous or syphilitic nature, are present. If the Wassermann test be negative, the pus from one of the softening nodules should be examined, and if the sporotrichum be present it will be demonstrated without difficulty by cultivation on Sabouraud's medium at room temperature. The diagnosis may be confirmed by the sporo-agglutination reaction, on the lines of the Widal test for typhoid fever, an emulsion of the spores of the sporotrichum being used instead of the bacillary emulsion.

If left undiagnosed, the disease, which is of a very chronic nature, will gradually extend. If detected early, it is readily amenable to treatment, yielding quickly to the administration of iodide of potassium. This drug should be given internally and also applied in a weak solution to the sores. Dr. de Beurmann states that in early cases and in otherwise healthy subjects cure may be expected in four to eight weeks.

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CHAPTER XXVIII

ASPERGILLOSIS

In the preceding chapters we have discussed the results of infection of the lung by various members of the streptothrix and sporothrix groups, parasitic organisms which belong to the Hyphomycetes or lowest order of mould fungi. We have now to consider the malady aspergillosis, pseudo-tuberculose aspergillaire, as it is termed by French writers, in which the malady is caused by mould fungi somewhat more highly developed and belonging to the family Perisporacidæ.[1a] The term "pneumonomycosis," or mould disease, sometimes used, would thus, strictly speaking, include all three diseases.

More than one member of the genus aspergillus have been found in the sputum and in the lungs in various diseases, such as pulmonary tuberculosis, bronchiectasis, and malignant disease. The presence of the parasite is generally noted only towards the end of the illness, and is not associated with any distinctive symptoms. Such cases of *secondary* aspergillosis, as they are called, consequently possess but little practical importance.

Of greater interest is *primary* aspergillosis, which is usually produced by the action of the Aspergillus fumigatus. This variety of the disease, as proved by Dr. Rénon,[2] to whose writings we are much indebted, is met with chiefly among the pigeon-feeders and hair-combers in Paris, who use in their work grain and flour, which are apt to be contaminated with the spores of the organism.

Clinically the malady may present various forms. It may sometimes resemble an acute broncho-pneumonia, and terminate fatally in two or three months, the lungs after death showing numerous patches of consolidation, in many places broken down into small cavities. Or, again, as in the case

reported by Drs. Arkle and Hinds,[3] in which the mycelium was found for the most part growing "in the walls of the alveoli and in the substance of the lung itself," it may present the signs and symptoms of a rapidly progressing emphysema with alarming attacks of dyspnœa, in one of which the patient referred to died. Two cases of membranous bronchitis of a very chronic type have also been traced to the presence of the parasite (Obici,[4] Rénon and Devillers[2c]).

Most commonly, however, the disease presents symptoms and signs which, like its pathological lesions, strongly resemble those of pulmonary tuberculosis. It may thus set in with cough and hæmoptysis, loss of weight, impairment of appetite, and increasing debility. Night-sweats also occur, and fever is often present, the temperature reaching 100° or 101°. The sputum is muco-purulent. On examining the patient, slight impairment of note at one or other apex may be observed, with blowing breathing. Later on signs of softening and cavity formation become apparent, as the consolidations break down. The course taken by this form of the disease is usually a chronic one, sometimes extending over several years, periods of quiescence alternating with those of activity. Recovery is by no means unknown, arrest being brought about by a form of fibrosis, as in tuberculosis of the lungs.

An interesting example of this variety of the disease was recently observed by Dr. Holden,[5] of Denver, Colorado, although in this case the parasite was the Aspergillus nidulans, which differs morpholgically from the Aspergillus fumigatus in the branching of its sterigmata. The patient was a married lady, aged forty-seven. Her symptoms, cough, sputum, loss of weight, clubbed fingers, and hectic fever, suggested phthisis, and the physical signs, commencing at the right apex and spreading downwards and attacking to a less extent the left apex, supported this diagnosis. Tubercle bacilli were, however, never found, and later the mycelium of the Aspergillus nidulans was discovered in the sputum. She died, greatly emaciated, after an illness lasting two years. An interesting feature of the case was the enlargement of the cervical and axillary glands, which at one time led to a suggestion of Hodgkin's disease. On section one of the glands was found to show no evidence of glandular structure, but to be con-

verted into fibrous tissue, between the fibres of which the parasite could be seen in large numbers on microscopical examination.

Diagnosis.—From what we have said it will be evident that the disease cannot be diagnosed during life by any clinical examination. Its recognition must depend upon the discovery of the parasite in the sputum. This may sometimes, as in a case recorded by Dr. Solmersitz,[6] be detected by the naked eye in the form of little masses, the size of a pin's head, resembling the grains met with in streptothrix disease, and, like the latter, consisting of a feltwork of branching mycelium. But more commonly it is first discovered in specimens of the sputum stained with such simple dyes as methylene blue, thionin, or safranin, when it appears in the form of branching mycelial threads, with spores scattered in and around. The threads have somewhat thicker walls, and are more irregular in shape than those usually met with in streptothrix infection, which they otherwise closely resemble. The aspergillus may sometimes also be demonstrated in unstained sputum which has been spread upon a slide, and treated with a solution of 20 per cent. potash.

To identify the parasite thus discovered, cultures from the sputum, expectorated into a sterilised vessel, must be made. For this purpose Sabouraud's agar medium (containing 1 per cent. peptone and 4 per cent. crude glucose), on which the aspergillus grows well at room temperature, may be used, or Raulin's medium,[2b] an acid liquid containing sugar, which, while favourable to the growth of mould fungi at 37° C., is inimical to that of ordinary bacteria. In this medium, if Aspergillus fumigatus be present, a mycelial growth will be noticed on the second day, starting from the particle of sputum introduced. A few days later this will reach the surface of the liquid, when it will quickly give rise to a whitish velvety veil, upon which after a few hours spores will develope, when its colour will change to a bluish-green, and later to a brownish-black. This colour of the culture will serve to distinguish Aspergillus fumigatus from other members of the genus, which have also been met with in sputum. To complete the identification of the organism intravenous injections of the spores must be made, preferably into the vein of a rabbit's ear. If the parasite be the Aspergillus fumigatus,

the animal will succumb in six to eight days with general dissemination of the fungus.

Should the patient die, the parasite thus discovered in the expectoration will also be found in the lung, unless the case be a very chronic one, scattered through the foci of disease, sometimes appearing as small white or brownish patches visible to the naked eye. In addition to the mycelium, spore-bearing hyphæ, with spores still attached, may also be discovered in the bronchi and alveoli.

Treatment.—No specific drug treatment is known, and the patient's resistance must be strengthened by abundance of fresh air and good food. Symptoms must be met as they arise. Cod-liver oil has been found of great value, and arsenic and iodide of potassium should be tried, since they have been found by Rénon to lengthen the duration of life in animals experimentally inoculated with the disease. In the less acute cases much may be hoped from such measures of treatment, and recovery may result.

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CHAPTER XXIX

PULMONARY TUBERCULOSIS

Ætiology.

Of the prevalence of phthisis and its importance to the community there can be no question. The statistics of the Registrar-General for England and Wales show that for the year 1918, the last year available, 46,077 persons died from it, this figure representing 7·6 per cent. of the total mortality from all causes. It should be further noted that this mortality from phthisis constituted 79 per cent. of the deaths from all forms of tuberculosis (58,073), thus showing the preponderant importance of the pulmonary form of the disease.

There is evidence to show that *tuberculosis* has existed amongst the people of very ancient civilisations. In a report on the early dynastic burials of Egypt, Professor Elliott Smith[1] and Dr. Derry describe two graves as containing skeletons which present evidence of tuberculous caries. One grave contained a man and a woman, the man with extensive caries of the three lower dorsal vertebræ causing acute kyphosis; the woman with an abscess cavity involving the bodies of the first and second segments of the sacrum, with extensive erosion of the bones. In the second grave, containing two men and a boy, one of the men had destructive caries of the tenth and eleventh vertebræ, with acute curvature; in the boy, the lower three dorsal and upper two lumbar vertebræ were affected. The dates of these early Egyptian burials were about 2,500 to 1,500 B.C. Professor E. Smith and the late Sir A. Ruffer[2] have also described in detail an example of Pott's curvature in an Egyptian mummy of the twenty-first dynasty, about 1,000 B.C., with spinal curvature and psoas abscess. No micrococci or tubercle bacilli could be found. These authors also describe a typical example of hip disease in a child which was probably

tuberculous. Another similar case was found by Dr. Wood Jones,[3] in which the lumbar region and left elbow-joint were affected. In the Nubian collection of bones in the museum of the Royal College of Surgeons there are two specimens[4] presenting all the characters of tuberculous disease of the spine, the skeletons being of dates about 3,000 and 2,000 B.C. respectively. Mr. Shattock, Pathological Curator to the College, who has been kind enough to give us the above information and references, states that in none of the specimens has it been possible to demonstrate the tubercle bacillus, but that the lesions in question can only be attributed to tuberculous disease.

Tuberculosis of the lungs has been known from very early times. Hippocrates first applied to it the term "phthisis," and a description of its clinical manifestations may be found in his writings and in those of Celsus, Aretæus, and Galen. The prevalence of the disease in these early times has not been recorded, but it must have been considerable.

The malady may be defined as one in which the affected tissues are invaded by a parasitic organism, the tubercle bacillus, under the influence of which they undergo specific alterations, become inflamed, and perish. It is a disease in which, through successive periods of activity, of toxic and suppurative fever, the body wastes and its functions languish. The terms "phthisis" and "consumption," so long applied to it, are thus appropriate, and may continue to be used, provided they are restricted to those forms of lung disease which are produced by the agency of the tubercle bacillus. They are clinical terms, as cancer is a clinical term, and are strictly applicable to the lesions and clinical phenomena brought about by pulmonary tuberculosis, just as cancer is applicable to the lesions and phenomena of carcinomatous or sarcomatous growth, and in this restricted sense we shall employ them synonymously in the course of this work.

In the first half of the eighteenth century Silvius recognised the anatomical relations between the nodular lesions of phthisis and their subsequent destructive changes, but it was Bayle who, in the latter part of that century, first described the tubercular nodule, and under that name recognised its constitutional character, its specific caseous changes, and its distribution in other organs than the lungs. It was not, how-

ever, until the specific cause of the disease was demonstrated by Koch in 1882 that its ætiology could be discussed on scientific lines. Hence, although in the ætiology of pulmonary tuberculosis we have to consider (1) predisposing causes—those causes or conditions which bring about a state of receptivity, or, in other words, a readiness on the part of the tissues to receive and harbour the tubercle bacillus—(2) the exciting cause, the *Bacillus tuberculosis* itself, it will be convenient to take the second heading of the subject first.

The Bacillus Tuberculosis.—The specific nature of tubercle, which was believed in by Laennec and by others long before his time, may be said to have been first demonstrated pathologically by Buhl in 1857, when he pointed out that an outburst of tuberculosis was almost always attributable to the previous existence of caseous matter somewhere in the body. Villemin[5] advanced the question a step farther in 1865, by showing that caseous matter introduced into a healthy animal produced tuberculosis, and that therefore the tuberculosis virus dwelt in caseated products. The memorable announcement of Villemin's discovery by Mr. (Sir John) Simon from the Chair of the Pathological Society of London was followed by numerous experiments in this country, the general result being that Villemin's conclusions were substantiated.[6] Fresh point and precision were given to the conclusions of Villemin by Koch's discovery, in 1882, of the causal agent in the transmission of tubercle, the *Bacillus tuberculosis*, which is present in every case of tubercle, which, when inoculated into susceptible animals, will reproduce the disease, and which, when freshly cultivated from the infected tissues of such secondary inoculation, will still, when reinoculated, produce the same disease.

In considering the nature and modes of access of this organism, it must in the first place be admitted that, although for an indefinite period in the ætiology of human and probably in that of bovine tuberculosis the incidence of the disease has been through the reception of bacillary infection derived from human or bovine sources, yet it cannot be doubted that the original or root source of the infection has been a saprophytic fungus, the common parent of all varieties of tubercle bacilli. Whether this fungus—probably a streptothrix—is now discoverable, and may still be a source of fresh infection amongst

cattle and, through them or independently, of other beings, is a question for botanists and bacteriologists to solve by further research.

The main facts of the life-history and potentialities of the tubercle bacillus may be summarised as follows:

1. The bacillus, whether occurring in the sputum, the tissues, or derived from cultures, appears as a rule in the form of minute rods 1·5 μ to 3·5 μ in length (about a quarter to half the diameter of a red blood-corpuscle) and 0·3 μ in breadth. They may be straight, but often they are somewhat curved. When stained, they either take the dye uniformly, or, as frequently happens, present a beaded appearance, portions of the bacilli taking the colour well, while the intervening parts remain unstained. Koch himself regarded these unstained portions as spores, and experiments in regard to the thermal death-point of the bacillus, which has been shown to vary between 65° C. and 90° C., support this conclusion, the spores, however, resembling those of a mould rather than of a fission fungus.[7]

The researches of Metchnikoff, Coppen Jones, and others have, however, shown that sometimes in the body-secretions, and always in old agar cultures, certain of the bacilli become much elongated and filamentous; and show *true branching*. Further, in not a few cases the terminal ends of these long forms, or of their branches, present definite *club-formation*. These appearances suggested to Metchnikoff that "the bacillus, as ordinarily met with, is not the end-stage, but only a stage in the developmental cycle of a filamentous fungus." This view has received much support from the work of Babes and Levaditi, who injected tubercle bacilli into the subdural space of rabbits, and found that after thirty days the foci presented appearances closely resembling those met with in actinomycosis, a central mass of bacilli and filaments being observed, surrounded by a radiating zone of clubs. Similar results have been obtained by Friedrich and by Schultze, and it would seem, therefore, that the organism should be regarded, not as a simple bacillus, but as a member of the streptothrix group belonging to the Hyphomycetes or mould fungi, which we have considered in a former chapter. With this view the thermal death-point of the tubercle bacillus, to which we have alluded, is in agreement.

2. The tubercle bacillus differs from the majority of pathogenic micro-organisms in not staining by ordinary methods. The method introduced by Ehrlich is that usually followed, and depends upon the fact that, though the tubercle bacillus takes up stain with considerable difficulty, yet the stain, once received, is held with tenacity. Accordingly, to stain the organism strong dyes are necessary; while, to decolourise the surrounding tissues and other bacteria, strong acids are permissible. To denote this unusual resistance on the part of the bacillus to the decolourising action of strong acids, the term "acid-fast" (*sauerfest*) has been introduced. The observations of Drs. Bulloch and Macleod[8] have shown that the peculiar staining qualities of the tubercle bacillus are dependent upon its containing a substance having all the chemical properties of wax, and, further, that its acid-fastness is due to the alcoholic ingredient of the wax, and not to its fatty acid element.[8(3)]

The method of staining as generally performed at the present day is as follows: The material to be examined—and in the case of sputum one of the small white points of caseous matter should if possible be selected—is spread out upon a cover-glass, dried, and then stained by Ziehl-Neelsen carbol-fuchsin (concentrated alcoholic solution of fuchsin, 11 c.c.; 5 per cent. aqueous solution of carbolic acid, 100 c.c.). The staining solution must be warmed until steaming occurs, and should be allowed to act for five minutes. The specimen is next washed in water, then placed for a few seconds (five to ten) in 20 per cent. hydrochloric acid. When no further colour is discharged, it should be transferred to 70 per cent. alcohol. After a few seconds, if no further decolourisation takes place, it is washed in water, counter-stained in Löffler's methylene blue (ten to twenty seconds), washed once more in water, dried between blotting-paper, and finally mounted in Canada balsam. Stained in this way, the tubercle bacillus acquires a bright pink colour, and is sharply differentiated from the surrounding blue tissues.

It should be stated at once, however, that this method of staining is not absolutely distinctive. The smegma bacillus and the bacillus of leprosy have long been known as acid-fast; and more recently numerous other organisms have been found to stain in exactly the same manner as the tubercle

bacillus. Amongst these we may mention certain varieties of streptothrix: the Timothy-grass bacillus and the bacillus, discovered by Möller on certain grasses having a wide distribution in nature; the "mist bacillus," found in the dung of various animals; and the butter-bacillus of Petri and Rabinowitsch. From tubercle the smegma bacillus may often be distinguished by its discolourisation by alcohol subsequent to its treatment with acid, in accordance with the routine method of staining which we have recommended, as well as by certain morphological differences. Of the others mentioned, the leprosy bacillus is met with but very rarely in this country, the acid-fast streptothrices would be distinguished by their branching, while the Timothy-grass bacillus and its allies occur only most exceptionally in sputum or other discharges from the human subject. Hence, for practical purposes, we may still rely upon staining methods in clinical diagnosis; but it must not be forgotten that, if scientific accuracy be required, the ultimate proof that the organism in question is the tubercle bacillus must rest, not upon morphology and staining properties, but upon cultivation and animal experiment.

3. The organism can be grown, although slowly, at a temperature of 37° C. to 38° C. (98° F. to 100° F.) on blood-serum; it can also be cultivated in broth, upon nutrient agar, and upon potato, provided 6 per cent. of glycerine has been added. In milk too the bacillus grows well (Klein). Another medium more recently introduced is Dorset's egg medium, and upon this, as shown by Dr. Stanley Griffith,[9] it may be cultivated with comparative ease direct from sputum, provided an emulsion of the sputum be first treated with antiformin, which destroys the saprophytic organisms gathered during the passage of the expectoration through the mouth. Gelatine is not suited to the cultivation of the bacillus, since, under ordinary conditions, it does not grow at a temperature below 29° C. (84° F.), though on glycerine-potato-broth growth has been observed at 22° C. For the characters of the growth on various media, we must refer our readers to works on bacteriology.

4. The bacillus is, under many conditions, of very tenacious vitality. When present in the dung of cows spread upon pasture-land, it has been shown by Dr. Stenhouse Williams[10] to retain its viability and virulence for at least five months,

and for twelve months when the dung is stored in a dark cellar. In decomposing sputum it preserves its virulence and capacity for development for six weeks or longer, and for six months if the sputum be allowed to dry. It is rapidly killed, however, in from a few minutes to some hours, according to the thickness of the film, by exposure to direct rays of the sun, and in diffused daylight it can only live for a few days. Exposure to ultra-violet rays is even more speedily fatal to its vitality.

Again, whilst the bacillus will resist a dry heat at a temperature of 100° C. for an hour, exposure for fifteen minutes to a moist heat of 90° C. or even less will destroy it. If suspended in water or milk, it will be killed by raising the temperature to boiling-point. On the other hand, it is capable of resisting very low temperatures, contact with liquid air (−186° C.) during a period of forty-two days having been shown by Swithinbank[11] to have little or no effect upon its vitality, though the virulence of the culture appeared to be slightly diminished.

Certain antiseptics are deadly to it. It is killed in less than a minute in a 5 per cent. solution of carbolic acid, and formalin and other antiseptics are also fatal to its vitality. Many substances can inhibit in culture the growth of the organism, but no antiseptic has as yet been found capable of arresting tuberculosis when administered to animals already suffering from the disease. As we shall have occasion to remark, however, when dealing with treatment, a drug which may not be directly fatal to the organism may indirectly so far affect it as to render it an easier prey to the vital forces of the host.

To the toxines, whether intra- or extra-cellular, which are formed by the tubercle bacillus in the course of its development or multiplication, many of the symptoms and most of the pathological features of the disease are attributable; but it cannot be said that at present we have any very exact knowledge of their chemical composition.

Tuberculosis is often met with in certain domesticated animals. In cows and pigs it is common, and in cattle the disease is known by the name bovine tuberculosis or *Perlsucht*. In the cat and dog it is not infrequent, and it occurs, though more rarely, in the horse, sheep, and rabbit. The guinea-pig, when inoculated, proves extremely susceptible; the common

fowl and other birds are frequently attacked. The bacilli which cause these widespread lesions in the animal kingdom present certain peculiarities among themselves, and may be differentiated into three types: the *avian*, *bovine*, and *human*. The bacillus of avian tuberculosis, though morphologically resembling very closely the human bacillus, differs sharply both from the bovine and human types in cultural characteristics, the temperature at which it thrives best, as well as its infectivity to animals. Man is but little susceptible to bacilli of the avian type, although in a few cases such organisms appear to have been isolated from tuberculous lesions in the human subject. Bacilli of the bovine variety are somewhat shorter and plumper than those of the human type, and show a much greater virulence when inoculated into the ox, the calf, or the rabbit. They are also capable of infecting the human subject, especially in early life, and bacilli having all the characteristics of the bovine type are not infrequently found in the tuberculous lesions of childhood, notably in the cervical glands, the lesions of abdominal tuberculosis, and those of alimentary origin. Infected milk and meat must, therefore, he regarded as a source of danger, though the bovine bacilli which they contain are probably less virulent for man than those of the human type, which are responsible, as we shall see, for nearly all cases of pulmonary tuberculosis.

We may now briefly summarise our knowledge respecting the distribution of the organism in the lesions of phthisis, a subject investigated by numerous observers, and notably by our colleague Dr. Percy Kidd.[12]

1. The bacilli are to be found in the sputa at some period of the disease in all cases of pulmonary tuberculosis (Plate XXVI., Fig. 1).

2. They are best seen in lung tissue which is undergoing rapid caseation, and may generally, therefore, be demonstrated with ease, and often in large numbers, in cases of acute caseous pneumonia or phthisis florida, and in the caseating walls of recent cavities, whether large or small (Plate XXVI., Figs. 2 and 3). They may sometimes also be found in abundance in the pus from empyemata of tuberculous origin (Plate XXVII., Fig. 2).

3. In other tuberculous consolidations of the lung the

PLATE XXVI

TUBERCLE BACILLI IN SPUTUM AND CAVITIES

FIG. 1.—Sputum from a case of pulmonary tuberculosis showing bacilli. (× 400.)

FIG. 2.—Naked-eye appearance of a cover-glass on which secretion from a pulmonary cavity was spread and treated by Ehrlich's method of staining. The tubercle bacilli are grouped together so thickly as to be visible to the naked eye as red points.

(Natural size. Preparation and drawing by Dr. Percy Kidd.)

FIG. 3.—Section through wall of a minute pulmonary cavity; inner margin of cavity teeming with tubercle bacilli; none elsewhere.

a. Cavity.
b. Bacillary margin.
c. Surrounding caseous tissue.

(From a preparation by Dr. Percy Kidd. × 75. This figure appears also in the *Transactions of the Royal Medical and Chirurgical Society*, 1885, vol. lxviii., p. 114.)

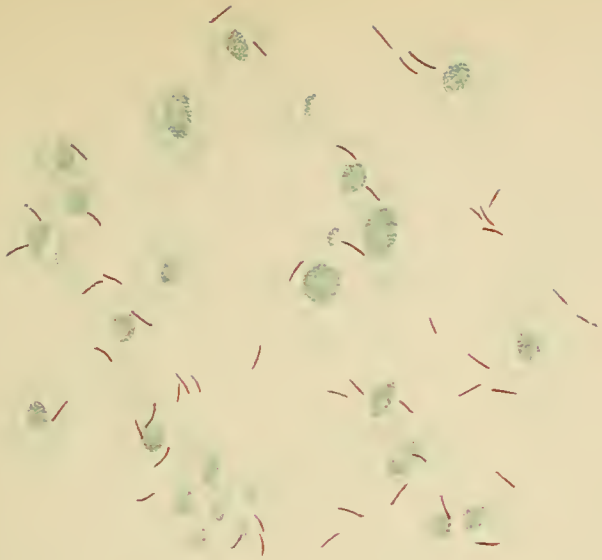

Fig. 1.

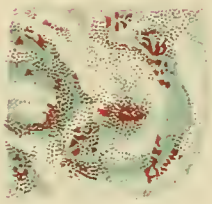

Fig. 2.

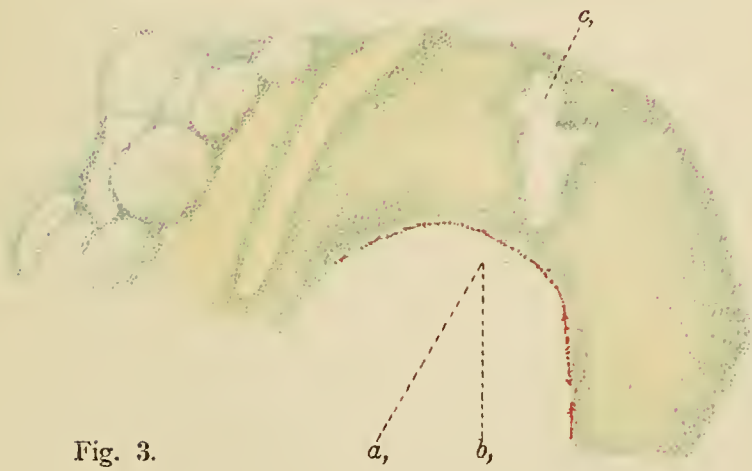

Fig. 3.

Tubercle Bacilli in Sputum and Cavities.

bacilli are often sparse and rather difficult to find. Large fields may be traversed without discovering them. Their presence, however, cannot be doubted, for this material, when inoculated, is virulent in producing tuberculosis.

4. In the granulations of miliary tuberculosis in the human subject bacilli are very generally, but not invariably, to be found, and often only in small numbers (Plate XXVII., Fig. 1).

5. The bacilli may sometimes be found in the blood in advanced cases of the disease, and occasionally even in the early stages.[13] They are frequently also to be discovered in the stools,[14] whether from swallowed sputum or intestinal lesions.

Channels of Infection.—The virus of tubercle may enter the body by infection of the ovum (congenital infection), or through the skin (inoculation), the food-passages (ingestion), or the air-passages (inhalation).

1. *Congenital Infection.*—It has been urged by Baumgarten that phthisis is usually acquired by direct infection of the ovum, the bacillus being conveyed to it by means of the spermatozoa, or more commonly through the blood of the mother, traversing the placenta and thus reaching the fœtus. Dr. Cobbett,[15a] in his work on the causes of tuberculosis, concludes that congenital infection is of more frequent occurrence than is commonly supposed.

Fœtal tuberculosis visible to the naked eye is rare, whether in men or animals, and experiment has shown that in the great majority of cases tubercle bacilli are absent from the tissues of the fœtus, though more extended observations in regard to stillborn children of tuberculous parents are required. The new-born child rarely reacts positively to the von Pirquet[16] tuberculin test. Further, the view of congenital infection presupposes a latent period, often of many years, before the germ takes on active growth and symptoms of tuberculosis arise. The theory may be accepted, as Dr. Sitzenfrey[17] has shown, as an explanation of certain cases of phthisis occurring in early childhood, but it cannot be held to explain the general incidence of the disease, either in children or the adult.

2. *Inoculation.*—Only very rarely can phthisis in man be attributed to this cause. Laennec himself, however, is said to have been infected in this manner, and similar cases are

recorded from time to time.[18] At the Brompton Hospital the following case occurred:

H. M., aged sixty-two, for twenty years pathological assistant, whilst staining some sputum in June, 1900, ran a pen covered with virulent expectoration into the flexor tendon sheath of the middle finger of his right hand. Tuberculous tenosynovitis resulted, but the trouble was arrested by the timely scraping out of the disease within two months of the primary infection.[19] He then returned to his work, and remained well until the spring of 1904, when he lost weight, developed cough and malaise, and, on examining his sputum, found tubercle bacilli therein. His chest was examined by one of us, and slight impairment of note was discovered at the left apex, but no adventitious sounds. He was admitted into hospital, and later went to the Brompton Hospital Sanatorium at Frimley, in Surrey. After some months' treatment he lost his cough, the bacilli disappeared from the sputum, and he was discharged with the disease arrested. There was no recurrence, and he lived comfortably, on a pension, until he developed epithelioma of the larynx, of which he died in March, 1915, at the age of seventy-seven.

In this instance it would seem probable that at the time of the injury a small focus of tuberculous disease formed in the lung, and that later, when his resistance diminished, it took on fresh activity, and made its presence apparent. Such cases are, however, quite exceptional.

3. *Inhalation and Ingestion.*—Until recently it was always believed that inhalation was the main channel of infection, ingestion playing quite a subsidiary rôle. The reasons for this belief were as follows:

(*a*) Numerous observers, Koch himself included, showed that pulmonary tuberculosis could be produced in animals by the inhalation of pure cultures of tubercle bacilli, or of pulverised sputum from phthisical patients. Bruno Heymann,[20] further, has actually demonstrated, by staining, the presence of tubercle bacilli in the pulmonary alveoli of guinea-pigs killed two hours after an experimental inhalation. This observation, we may add, derives confirmation from the work of Ballin,[21] who, experimenting with the spores of *Aspergillus fumigatus*, proved their presence microscopically in the alveoli after half an hour's inhalation, and within three hours they were found penetrating the alveolar walls.

(*b*) The view received support also from the fact that enormous numbers of bacilli may be present over long periods

PLATE XXVII

TUBERCLE BACILLI IN ACUTE MILIARY TUBERCULOSIS OF LUNG, AND IN PUS FROM A TUBERCULOUS EMPYEMA.

FIG. 1.—ACUTE MILIARY TUBERCULOSIS.—Lung: two alveoli which have become fused together; alveoli filled with large epithelioid cells; tubercle bacilli in large numbers between the cells, and in a few instances within the cells (a.a.).

(From a preparation by Dr. Percy Kidd. ×400. This plate appears also in the *Transactions of the Royal Medical and Chirurgical Society*, 1885, vol. lxviii., p. 114.)

FIG. 2.—EMPYEMA OF TUBERCULOUS ORIGIN.—Pus removed from the interior of the pleura, showing abundance of bacilli.

(From a preparation by Mr. H. H. Taylor, F.R.C.S. ×400.)

Fig. 1.

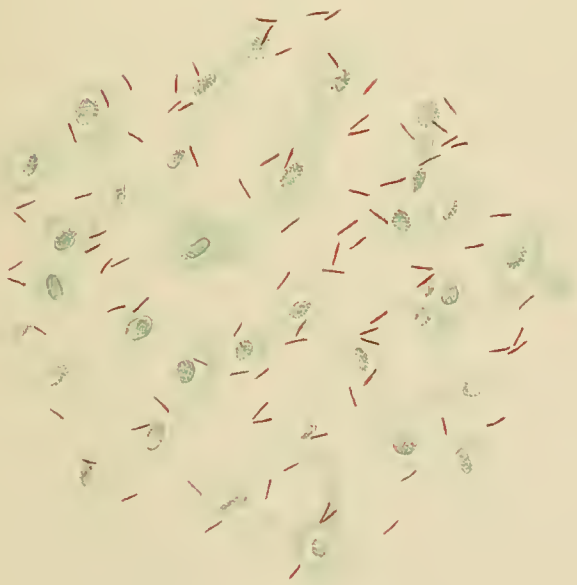

Fig. 2.

Tubercle Bacilli in acute Miliary Tuberculosis of Lung and in pus from a Tuberculous Empyema.

of time in the sputum of a consumptive patient, in some cases, as computed by Professor Cornet, reaching the total of 7,200,000,000 a day, although a certain proportion of them may be no longer living. It seemed most reasonable to believe that some of the organisms thus so profusely ejected find access to other persons by means of the respiratory tract.

(*c*) Birch-Hirschfeld, Schmorl, and others demonstrated that in very early cases the primary lesion was sometimes found in the walls of the fine bronchi, whence it spread by aspiration to the alveoli.

The above facts are strongly in favour of the view that the sputum is the chief source of danger in the spread of phthisis, and that the main source of infection is by inhalation. Both Koch and Cornet believed, and with them Köhlisch's[22] more recent experiments are in accord, that the mode of this infection is by discharged sputum becoming dried and mingled with dust, the dust thus contaminated being wafted into the air by sweeping, traffic and winds, and in this way becoming inhaled. Cornet disbelieves in moist sputum as a cause of phthisis. Professor Flügge, of Breslau, and his pupils, on the other hand, maintain that the infection more frequently arises from the direct inhalation of the fine droplets of infected sputum which, as they have shown, are scattered into the air by the consumptive patient in loud speaking, and more so in coughing and sneezing. Seeing that such droplets may remain suspended in the air as long as five or six hours, and as Dr. M. H. Gordon[23] has shown, may be conveyed to great distances, in one case as far as 71 feet from a person speaking vehemently, ample opportunity is given for their inhalation. In quiet reading or speaking, however, no such ejection appeared to occur.

It has recently been shown, however, by Dr. Chaussé[24] that such droplets, which consist mostly of saliva, contain but very few tubercle bacilli, and that experimentally it is difficult to infect guinea-pigs when exposed to such droplets by allowing patients to cough repeatedly into the box in which the animals are confined, and this when the expectoration of the patients was proved to contain an average of 60,000 tubercle bacilli in each milligramme of sputum. Of 79 animals so exposed, only one acquired tuberculosis. On the other hand, it proved easy in Dr. Chaussé's hands to infect guinea-pigs by the inhalation

of dried dust derived from the handkerchief used by a consumptive patient, if this, after partial drying for forty-eight hours, was shaken in the box containing the animals. Several handkerchiefs from different patients were used, and in all 41 guinea-pigs out of 73 exposed acquired tuberculosis. Without denying that in circumstances of great intimacy, as when two or more patients occupy the same bed, infection by droplets may become operative, the experiments quoted indicate that under ordinary circumstances dust infection, derived often from the patient's clothes, bed-linen and person, is the more important factor in the spread of the disease.

That inhalation directly into the air-passages, whether of sputum-infected dust or droplets, is not the sole or even the chief method of infection is, however, maintained by some observers on the following grounds: (*a*) It is well known that if suitable animals, such as guinea-pigs, calves or pigs, be fed with tuberculous material, lesions of the intestinal tract will result, with secondary involvement of the mesenteric glands and later of other organs of the body, among which the lungs may be included. Such involvement of the intestine often occurs in phthisical patients who swallow their sputum, and primary intestinal tuberculosis in children is no doubt also due to the ingestion of tuberculous matter. (*b*) The work of numerous observers also shows that under certain circumstances, without producing any local lesion, the bacilli may pass directly through the wall of the alimentary tract, or, as indicated by Dr. Walsham,[25] in his Weber-Parkes Prize Essay, through the tonsils to the lymphatic glands, and thence to other organs. Thus, in a case observed by Dr. Sidney Martin, in which a pig was fed on meat from a tuberculous cow, no lesion of the tonsil resulted, but the glands below the jaw on the right side were involved, and the lungs were also infected; the other organs remained natural. In analogous cases in man it is probable that the bacilli reach the lungs by means of the blood-stream, there appearing to be in the human subject at least no direct lymphatic connection between the cervical glands and the lungs or pleuræ.[26] In other experiments the mesenteric glands have alone been affected, the bacilli having passed through the intestinal wall without leaving any trace of their passage. It may be admitted, therefore, that the lungs are in some cases infected by bacilli which

have been swallowed, and have entered the system from the alimentary tract.

Von Behring's theory, which affirmed that pulmonary tuberculosis, though not congenital, originates commonly in early childhood through the ingestion of infected milk, the germ developing and the disease appearing only in adult life, is negatived by the fact that in nearly all cases, as we shall show, phthisis is produced by bacilli of the human, not the bovine, type.

Calmette and his pupils, among whom may be mentioned Vansteenberghe and Grysez,[27] following von Behring's theory, urge that infection through the alimentary canal is not the exception but the rule, and that phthisis does not result from the direct inhalation of tubercle bacilli into the lung, but that the organisms, whether in infected dust, droplets, or tuberculous milk, are swallowed, and then pass to the mesenteric glands, and so to the lungs. Calmette bases his conclusions, which he has voiced at various congresses, on experiments designed to show that phthisis, or tuberculosis confined to the lungs and thorax, can be produced by the introduction of tubercle bacilli into the alimentary tract, and that pulmonary anthracosis is not, as is commonly believed, the result of the inhalation of carbon particles, but is really of intestinal origin. His results, however, have not been confirmed by Dr. Cobbett[15b] and other observers, who show clearly that pulmonary anthracosis as well as phthisis are both in the great majority of cases the result of an inhalation infection, though it is not denied that in cases of alimentary infection the lungs may eventually become involved when the disease, after affecting the mesenteric glands, has become generalised. This conclusion is supported by a series of experiments by various observers, notably by Findel[28] and Alexander,[29] which prove that a much larger dose of bacilli is necessary to produce infection through the alimentary than the respiratory tract. We may conclude, therefore, that the old view is correct, and that in the ætiology of phthisis the main channel of infection is by inhalation, ingestion playing only a minor rôle.

Milk Infection.—The question of the derivation of human tuberculosis through milk or butter from infected cows is one of importance, involving as it does large farming interests and State measures of a preventive kind. In considering this

question, we must carefully separate from such cases those in which from dirty surroundings milk becomes contaminated by tuberculous dust, thus coming within the category of *contaminated foods.* The question now under discussion is solely concerned with milk from diseased cows, the organism of infection being the bovine tubercle bacillus, the milk thus coming within the category of *diseased foods*, such as tuberculous meat.

There have been two Royal Commissions dealing *inter alia* with the subject of tuberculous milk, and both reported strongly in favour of tuberculosis arising from its ingestion. Since the publication of the Report of the second Commission[30] in 1907, much work has been carried out in differentiating the bacilli, whether of human or bovine type, which are to be found in the various forms of tuberculosis, and we are now in a position to speak with some precision on this subject. In the first place, it has become clear that *fatal bovine infection in the adult is of extreme rarity, but that in children under five years of age it is not uncommon.* Dr. Cobbett,[15c] to whose work we are much indebted, calculates that about one-third of all fatal cases of tuberculosis in children under the age of five is attributable to a bovine source, presumably infected milk, and that in the year 1911 (the returns for which he used in his calculations) rather over 3,000 deaths could be traced in England and Wales to this cause.

This conclusion is based on the following evidence. Bacilli of bovine type have not hitherto been found in the pulmonary lesions of phthisis. In the sputum from nearly a thousand cases examined, in only four were bovine bacilli found, the organisms in all the rest being of the human type. It is clear, therefore, that *with but rare exceptions, pulmonary tuberculosis in the adult, which accounts at the present time for 79 per cent. of the deaths from all forms of tuberculosis, is produced by bacilli of the human type, and is the result of infection from human sources*, and in our efforts towards the eradication of the disease this cardinal fact must be borne in mind.

With regard to other forms of tuberculosis, according to Dr. Cobbett some 17 per cent. of cases of general tuberculosis (including meningitis) and 50 per cent. of cases of fatal abdominal tuberculosis are caused by bovine bacilli, both

occurring mostly in children. In tuberculosis of the cervical glands, which is not generally fatal, 50 per cent. of the cases in England would appear to result from a bovine infection, and perhaps 70 per cent. in Scotland, the incidence of such infection being much higher in childhood than in the adult. Of cases of bone and joint tuberculosis, one in five may be attributed in England to the bovine type of bacillus, and in Scotland a higher proportion, in each case the percentage in childhood being much greater than in the adult. From an analysis of the figures, Dr. Cobbett concludes that roughly 6 per cent. of all cases of fatal tuberculosis may be attributed to the bovine bacillus, such cases occurring for the most part in young children, and two-thirds of them being of the abdominal variety, including ulceration of the bowel, tuberculosis of the mesenteric glands, and tuberculous peritonitis. When we remember that in addition to these fatal cases much suffering and permanent crippling is caused in the non-fatal attacks, especially those of bone and joint tuberculosis, a considerable proportion of which are produced by the bovine bacillus, we see that the danger from infected milk, though of minor importance when we view the subject as a whole, is yet one by no means to be ignored. It is fortunate that it may be obviated by Pasteurising the milk or raising it to the boiling-point.

It is interesting to note that the ethnographical data collected by Dr. Bruno Heymann[31] support the conclusion thus arrived at on bacteriological grounds that the rôle played by infected milk in the dissemination of *pulmonary* tuberculosis is a negligible one, and that this form of tuberculosis is, in fact, spread, as we have urged, by the inhalation of infected material derived from human sources. This observer showed that in various countries, such as Greenland, the Gold Coast, and parts of Roumania, where milk cannot possibly be obtained, or where, as in Egypt, it is too dear for the natives to buy, consumption is nevertheless rife, and other forms of tuberculosis are often met with. Further, in the Faröe Islands and in Japan, tubercle of all kinds had long been frequent, though *perlsucht* has only recently been introduced.

The Question of Contagion.—There arises out of a consideration of the various methods of infection the grave question

as to how far pulmonary tuberculosis is a contagious disease, how far it arises from a person-to-person infection analogous to that by which the contagia of measles, smallpox, and other such diseases are most commonly conveyed. The belief by many physicians in the contagiousness of phthisis is not merely of laboratory origin, dating from the demonstration of the tubercle bacillus: it has existed throughout the history of medicine. In a sense it is true, in another sense it is greatly exaggerated. It is true in the sense that if the opportunities for infection are intimate and prolonged, and proper precautions against the spread of the disease are not forthcoming, so that *massive doses* are absorbed, as happens but too frequently in the overcrowded and insanitary dwellings of the poor, then direct infection may, and no doubt does, occur.

Of patients belonging to the lower middle and working classes admitted to the Brompton Hospital, only about one-third would appear to be aware of any history of personal infection, if we may judge from the 100 patients under the care of one of us, in whom this point was inquired into by Dr. Wijeyeratne. He found that 27 of these 100 patients gave a definite history of exposure to domestic, and 8 to extra-domestic infection, at some time previous to the onset of their disease, making a total of 35 per cent. In the remaining 65 no history of the kind could be obtained. In certain of the cases the exposure to known infection had occurred several years before, and it must not be forgotten that small tuberculous lesions, such as a caseous area in a lymphatic gland, or a quiescent nodule in the lung, may remain dormant for long periods, to break into active disease under conditions of anxiety and strain, and in this way the origin of cases of phthisis may sometimes be traced back to infection in earlier years of life.

It must be clearly understood, however, that the contagious character of the malady, thus admitted under certain circumstances, is of a totally different order from that met with in the common contagious diseases such as measles and scarlet fever. In the latter a short, even a momentary, contact with the patient is sufficient to convey the disease. In phthisis, on the other hand, the contact must be intimate, repeated, and prolonged to be effective, and even then, if the patient has been properly trained in the disposal of the sputum and

the use of the handkerchief when coughing, and regard had to cleanliness in the surroundings, and to proper ventilation and adequate nursing when the patient is bedridden, there is little or no risk of infection. In the upper classes tuberculosis is, as a rule, acquired rather by the inhalation of dust from public waiting-rooms, railway-carriages, trams, omnibuses and the like, in which tubercle bacilli derived from infected sputum have not infrequently been found.

One of us can, however, recall the case of a wealthy man suffering from consumption and living in a large house, whose dirty habits led to the infection of his two daughters, who both died of the disease, although his wife escaped. Similarly the habits in regard to spitting of the North American Indians, so graphically described by Dr. Quevli[32a] of Tacona, and of the natives of South Africa, with their ignorant disregard of all the rules of hygiene, explain the rapid spread of the disease among these people. Wherever, also, the disease is freshly introduced, there the standard of immunity ranges low.

It is interesting to note that the mouthpieces of public telephones, even when used by consumptive patients (as shown at the Brompton Hospital Sanatorium, at Frimley), have been found on investigation to be free from tubercle bacilli, and should not be regarded, therefore, as sources of public danger, provided they are kept properly clean.*

That the danger of personal infection is not great if proper precautions are taken is indicated by the fact that in *consumption hospitals* and large sanatoria the disease is not abnormally prevalent amongst the officials and servants. The observations of the late Dr. Theodore Williams,[33] upon the health of the staff of the Brompton Hospital showed that at this institution during the years 1846 to 1882—a period when open-air treatment was not practised, and when the ventilation of portions of the hospital was not all that could be desired—phthisis did not develope among the staff, whether physicians, assistant physicians, clinical assistants (house physicians), or nursing and general staff, some of whom had lived for many years in the hospital, more frequently than

* This statement is based upon reports by Dr. Klein, and more recently by Dr. Spitta, which have been courteously placed at our disposal by the Postmaster-General.

might be expected in any large institution not especially devoted to consumption, or, indeed, in any urban population. This conclusion was based upon the records of several hundred persons whose life-histories were traced for many years after leaving the institution by the then Resident Medical Officer, the late Mr. Vertue Edwards. More recently Dr. Theodore Williams[32b] published statistics of the medical staff of the hospital from 1882 to 1909, which yielded similar conclusions, though it should be noted that among the

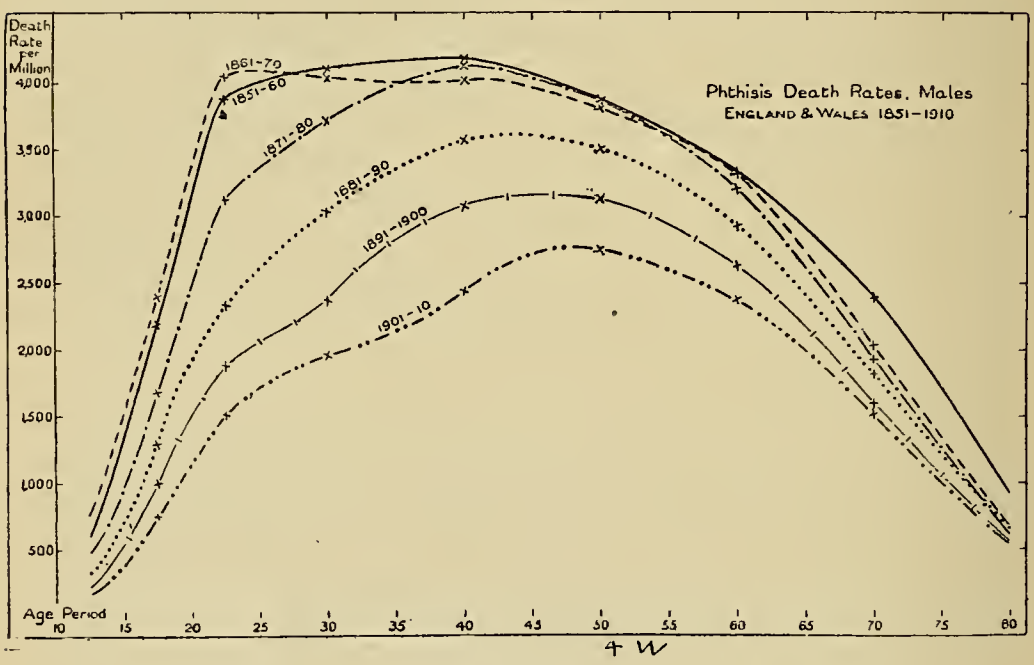

FIG. 42.

From Dr. Brownlee's Report to Medical Research Committee, 1918, diag. ii. In this and the following diagram (1) the general decline in death-rate from phthisis is shown in successive decades; (2) the curves also show the far greater decline at the earlier ages and the greater relative mortality, especially amongst males, between the ages of 35 to 55 in the later decades.

porters, whose duty it was to handle and sterilise the linen and sputum, two deaths from phthisis had occurred.

From these observations, which have been confirmed by the results from other consumption hospitals, it seems clear that the danger of personal infection is not a great one, provided that common-sense precautions are taken.

The observations of Sir Hugh Beevor[34] upon *the prevalence of phthisis in the rural districts of Norfolk* during three succeeding decades (1861 to 1890) are also interesting in this respect. The figures show that in each decennium the death-

rate from phthisis in these small rural populations (varying in number from 2,000 to 6,000) was in each remarkably constant, nearly always falling within 10 per cent. of the mean rate for that of all the rural districts, and never showing those marked irregularities which are met with in the case of the common epidemic or infectious diseases. The figures indicate, as Sir Hugh Beevor points out, that, given fairly satisfactory surroundings, the influence of case-to-case infection sinks in importance, other contributory or favouring factors playing the chief rôle. The steady line of decline of the tuberculosis

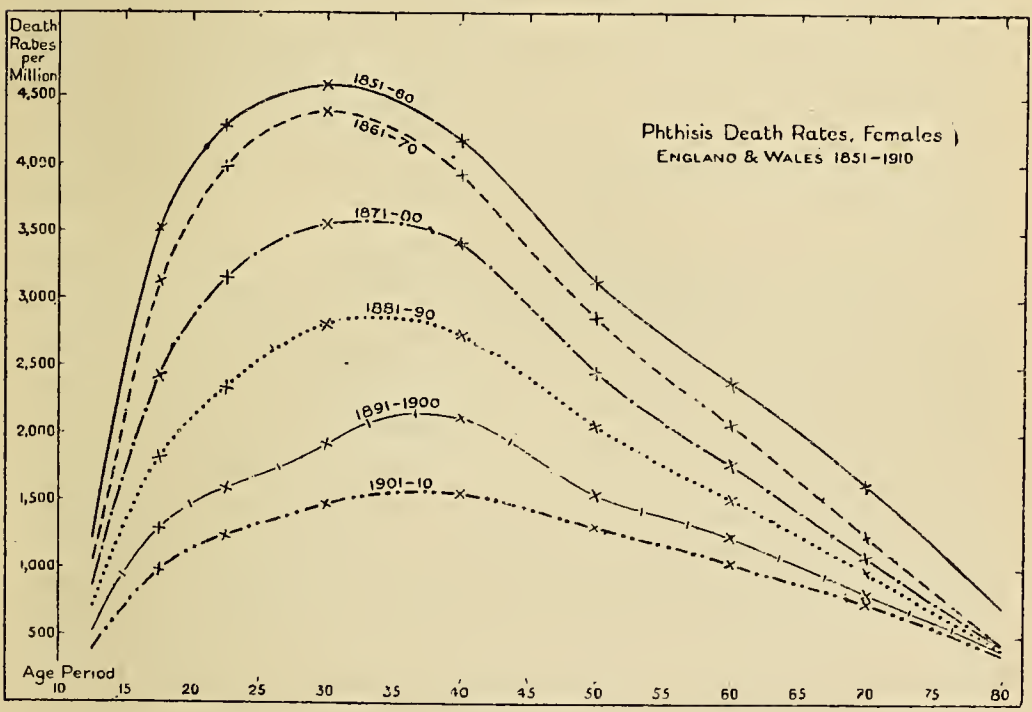

Fig. 43.

From Dr. Brownlee's Report to Medical Research Committee, 1918, diag. iii.

death-rate through successive decades (see Figs. 42 and 43) is further evidence that the contagious element in the malady no longer resembles, either in type or degree, that obtaining in the ordinary contagious diseases.

Marital or Conjugal Tuberculosis.—Evidence in favour of personal infection has been deduced from the cases in which the disease has been communicated from husband to wife, and *vice versa;* and the remarkable series collected by the late Sir Hermann Weber[35] of nine consumptive husbands who lost eighteen wives, one of these pathological Bluebeards being

responsible for four, another for three, four others for two, and three for one each, was very striking. Such dramatic experience is, however, quite unusual, and the exact conditions under which these people were living are not stated. Certainly in the upper classes, in our experience, conjugal tuberculosis is uncommon.

This view is, however, by no means generally accepted. Dr. Ernest Ward,[36] Tuberculosis Officer for South Devon, has recently reported a series of cases to illustrate the frequency of conjugal tuberculosis, amounting to over 50 per cent., in his district amongst the working and lower middle classes; but the associated conditions are not stated, and we can only conjecture the degree of overcrowding or general insanitation that could produce such results, and to which the parents of households would, of course, be exposed with some excess of intensity.

Statistics on larger lines are completely adverse to such restricted experiences, and when closely scrutinised suggest that any slight increase of vulnerability observed in such cases depends upon other factors besides mere contiguity. Dr. Longstaff[37] has pointed out that, seeing how prevalent consumption is, it must happen, in the ordinary course of events, that husbands and wives will die of the disease, not very infrequently within short periods of each other, without any question of infection arising. Basing his results on mathematical calculations deduced from the Registrar-General's Reports, he estimated that the prevalence of conjugal tuberculosis was not greater than might be expected as a matter of coincidence.

The late Dr. E. G. Pope, Professor Karl Pearson, and Miss Elderton,[38] also investigated the subject in an elaborate statistical memoir, and concluded that consumption in husband and wife does occur more frequently than can be explained by the doctrine of chances, and that there is some sensible but slight infection between married couples. On a further analysis of the statistics of Dr. Pope, comparing them with calculations of his own and of Miss Elderton and others engaged in his laboratory, Professor Pearson[39] concludes that for the middle and professional classes there is a definite marital relationship of persons with similarity of constitution and of physical and psychical attributes

which is due to unconscious selection in mating. To this disposition towards marriage manifested by persons having similar constitutions and attributes he gives the name "assortative mating," and he thinks that it may account for the slight excess of liability to infection which he finds to exist amongst married people. This relationship or correlation holds good for eye colour, general health, tone of voice, for certain neuroses, including insanity, and other features which cannot in any sense be regarded as of infectious origin. The index of similarity or correlation for married people of the same social status with regard to phthisis—that is, their relative vulnerability to that disease—is almost identically the same as that for the main features just named, and may be represented by the decimal 0·24 to 0·28. The index falls in both cases as we descend the social scale, being 0·16 for the prosperous poor and entirely negligible for the very poor. It seems clear, then, that the increased tendency of married couples to become both affected with phthisis, slight as it is, is not wholly attributable to direct infection, but also to an increased vulnerability to distributed or environmental infection. A similar conjugal disposition to insanity is to be noted, only with increased force—0·30; and to alcoholism with still greater intensity—0·7. Dr. Goring[40] concludes from his separate researches that "there is no evidence of marital infection." "The incidence of phthisis in both husband and wife noted by Pope, Greenwood, and others, may be due to assortative mating."

Another point of great importance in the incidence of phthisis as bearing upon the question of direct marital infection, and *a fortiori* of person to person infection generally, is the fact pointed out by Karl Pearson[39a] that the probability of the offspring of a tuberculous father or mother being tuberculous is far greater than the probability of a wife or husband being similarly attacked. "The father is twice as dangerous to the child as the husband to the wife! The mother is only very slightly more dangerous than the father at very early ages."

Heredity would thus seem to be the supplementary factor involved in assortative mating, which may account for the slightly increased number of instances in which husband and wife are both involved in tuberculosis. And Professor Pear-

son, in confirmation of this, finds that there is an appreciable excess or concentration of heredity to tuberculosis amongst the forebears of married people; the stocks from which they are drawn having a correlation in this respect of 0·30.[39b]

It is worthy of special note that amongst the very poor, where assortative mating practically does not obtain, tuberculosis is much more prevalent than amongst the middle and professional classes, the intensity of the environmental infection from overcrowding and the diminished resistance from adverse conditions of life already considered no doubt accounting for the difference, conditions which are intensified in the conjugal relationship.

If we accept the views put forward in the above sections their bearing on prevention is obvious. The consumptive patient *who is properly trained and willing and able to carry out instructions* is no real source of danger. Still less should such patients, who need our sympathy, be regarded as lepers to be shunned. On the other hand, the selfish or ignorant patient who disregards all precautions, or the sufferer who from increasing weakness, lack of proper nursing, and overcrowded surroundings is incapable of doing what is required, becomes a grave danger to his fellows. In such cases, voluntary segregation and the admission of the patient to a home or hospital is the line of treatment indicated. Sir Arthur Newsholme[41] draws an interesting parallel between the increase of institutional treatment and the decline of the phthisis death-rate during the latter half of the last century. An increased provision of beds for advanced cases is, however, still urgently needed in not a few localities. We shall further consider this question in the next chapter.

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CHAPTER XXX

PULMONARY TUBERCULOSIS—(*Continued*)

Ætiology—(*Continued*)

Let us now consider the conditions which bring about that aptitude for the reception of the bacillus which, in view of the manifold opportunities of infection, is of such paramount importance.

1. **Constitutional Liability.**—Apart from all other ætiological considerations, the constitutional liability to tuberculosis must be taken into account, this constitutional tendency being most purely and strikingly manifested in *hereditary*, less completely so in *acquired, liability*. We do not maintain the first, and it would be absurd to argue the second as being independent of the surrounding climatic and social conditions which we have yet to discuss; but when these have been allowed for, there still remains to be considered the personal element in the disease—the character of the *soil* as opposed to that of the *seed*—which in many cases exercises an influence which cannot be gainsaid.

The constitution of a man has been defined by one of us as "his build, the integrity or otherwise of the tissues of which each part of his body is made up, and the wholesomeness or otherwise of the juices with which they are bathed; the sum of his vital force, his cell-quickening power, which shall bear the call of judicious expenditure for a long or but a brief period of time."[1] This material and dynamic constitution is born with the infant, developed during the period of growth, and maintained with waning completeness during the wear and tear of subsequent life. Hereditary constitutional defect means unsoundness of original construction with regard to some organ or tissue at birth. Acquired constitutional defect means that some part of the human mechanism has suffered deterioration from deficient supply of the needs of growth

and function, through wilful or involuntary exhaustion of vital powers, or from imperfect recovery from acute disease.

Inherited Tendency to Phthisis.—In considering this question, it has been usual to quote statistics showing that, of those suffering from consumption, many have lost a parent or some near relative from the same disease, and to gauge the degree of danger from the resulting figures. Thus, in the first medical report of the Brompton Hospital, of the 1,010 patients investigated, a history of consumption in one or other parent (and sometimes in both) was obtained in 246 cases, or 24·4 per cent. Dr. C. J. B. Williams and Dr. Theodore Williams,[2] again, taking only their private patients, found that, of 1,000 cases carefully investigated by them, 120 gave a parental history of the disease, while in 484 some near relative had fallen a victim. The conclusion at which they arrived was that "direct hereditary predisposition" might be traced in 12 per cent. of the cases, and "family predisposition" in 48 per cent.; and this estimate, coming from the upper classes, who are protected from many of the more potent causes of the disease, they were inclined to accept as a fair statement of the influence of hereditary predisposition. Our own experience would be in accord with these results. One of us,[3] looking back upon the histories of 450 cases of consumption taken consecutively from case-books of a few years previously, found 208 cases with a definite history in the family, 123 with a negative history, and 119 in which the history was not recorded (most of these latter would be negative). Of the 208 cases with positive histories, in 43 several direct relations were affected (father and mother, 4; father and grandparents, 2; father and brother or sister, 5; mother and brother or sister, 11; two or more brothers and sisters, 20; brother and grandparents, 1). In 30 and 33 cases respectively the father or the mother was affected, yet in 4 instances only were both involved, and we should regard this as in excess of our general experience.

More complete data were collected from 383 patients at the Crossley Sanatorium, Delamere Forest, by Dr. W. C. Rivers, and were discussed in an interesting memoir by Professor Karl Pearson,[4] who, however, was careful to point out that the investigation was preliminary, and that his conclusions needed confirmation from more extended data. After a

detailed analysis of the figures by modern statistical methods, he concludes: "The diathesis of pulmonary tuberculosis is certainly inherited, and the intensity of inheritance is sensibly the same as that of any normal physical character (*e.g.*, stature, span, cubit, eye-colour) yet investigated in man. . . . A theory of infection does not account for the facts." He is further "inclined to think that the risks run, especially under urban conditions, are for tuberculosis, as for a number of other infectious diseases, so great that the constitution or diathesis means almost everything for the individual whose life cannot be spent in self-protection."

A reference to the paragraph on "marital infection" in the preceding chapter will bring to mind that the marital relationship, in the more intellectual classes at all events, by virtue of the unconscious sexual attraction of constitutional affinities, brings into close association persons with a somewhat stronger strain of hereditary predisposition to tubercle infection, and that amongst them there is therefore an increased vulnerability to any environmental conditions tending to tuberculosis.

Let us now ask ourselves how far Professor Karl Pearson's conclusion is supported by clinical experience. All physicians who have seen much of phthisis will be able to recall instances in which one member after another in a family falls a victim to the disease, and this after the individuals have grown up and separated widely. A striking example of this has come under our notice. A lady, the wife of a country gentleman in good position, died of consumption at the age of forty-eight, leaving fifteen children. Five of these (daughters) married and left home, and subsequently died of the same disease at the approximate ages of forty-eight, forty-nine, thirty, twenty-seven, and twenty-six respectively. In addition, one unmarried daughter developed consumption and died at San Remo at the age of nineteen, and one son developed phthisis in India, and died in Australia at the age of twenty-seven. We may add that at the date at which the facts were brought to our notice two grandchildren, aged thirty-three and eighteen (children of the daughters above referred to who died of phthisis at the ages of forty-eight and twenty-six respectively), had also developed consumption.

It will be urged by those who oppose the doctrine of heredity that the seven out of the fifteen children, who thus developed phthisis, acquired the disease because they were infected from their mother in comparatively early life, the organisms then remaining dormant for years. Such a proposition is not impossible in view of the fact that living and virulent tubercle bacilli may be found post-mortem in caseous and even calcareous foci in persons who during life had not wittingly suffered from tuberculosis.[5] Even, however, if we accept the correctness of the assumption, we have to ask why these patients eventually developed active pulmonary tuberculosis. Phthisis is an exceedingly common complaint, and the opportunities of infection under ordinary conditions of civilisation seem infinite. The observations of Naegeli show that of post-mortem examinations made between the ages of eighteen and thirty, 96 per cent. gave evidence of having been at some time infected, and that above thirty no individual is free: yet the disease does not develope save in a restricted percentage of the population.

It appears to us that the only answer lies in the doctrine of heredity, the inheritance, as it has been termed, of a "tubercular diathesis," rendering the soil of certain family constitutions more suitable for the growth of the bacillus than that of others, so that, when infected with a suitable dose of the virus, such individuals succumb when their more fortunate brethren escape.

This doctrine is no fantastic one, and is only what we might expect if we consider the matter from a biological standpoint. The bacillus is a member of the vegetable kingdom, and it is notorious how selective such organisms are. As Sir James Crichton-Browne has truly said: "The seed of certain plants will grow only in a clay soil, while those of others will sprout only in sand or chalk; and mushrooms, which are not remotely allied to the fission fungi or bacteria, are nice in their taste, and refuse to increase and multiply on an exhausted bed or one of unsuitable material; while they flourish copiously when their spawn is spread on an appropriate nidus." So it is with the tubercle bacillus, which exhibits this same selectiveness. It refuses to grow in broth or on agar-agar, and yet, if a little glycerine be added, growth at once takes place. That some slight modification of the soil in certain families should favour

or inhibit the growth of the organism is to us, therefore, not surprising.

If this doctrine be true, we should expect that the different races of mankind, following the example of the animal kingdom, would exhibit a different liability to disease. There is some evidence to show that this is so, though here again it is difficult to separate the race factor from the other elements in the causation of the disease. It is admitted, however, in America that the negro is very liable to consumption, and that, when acquired, it runs in him a rapid course. Among the Jewish race, on the contrary, the mortality from phthisis, both in London and New York, as in most of the great cities of Europe, is shown by statistics to be less than that of the native population, and it is not impossible that this lower death-rate is due in part at least to diminished racial proclivity.[6] Major Johnston, of the Indian Medical Service, has also shown that among the races which compose the Indian Army there is a marked variation in the incidence of pulmonary tuberculosis, ranging from 73 per 10,000 among the Gurkha soldiers to 17·8 and 15·0 per 10,000 among the Sikhs and Mahrattas respectively. He considers that the racial factor plays a definite share in this varying liability.[7]

It is thus clear that inheritance is an important factor in the ætiology of phthisis, and that it manifests itself in a special idiosyncrasy of the tissues in certain families and races whereby they become more than usually favourable to the growth of the tubercle bacillus. It is only right to add that the effect of this factor is to tend to eliminate the more susceptible, and in the course of generations to increase immunity amongst the survivors.[8]

2. **Climatic Causes.**—Phthisis flourishes, as we have said, in every climate, and it is not possible to point to any country which has a monopoly of the disease. True, the death-rate from consumption differs much in different regions, but this would seem to depend in great part not so much upon varying meteorological data as upon other factors which favour the incidence of the disease. Wherever the population is scattered and leads an open-air life, there the disease is less frequent. Wherever overcrowding is rife, and the evils of civilisation are apparent, there it is more common. There are, however, certain climatic factors which deserve consideration.

(*a*) *Altitude.*—Evidence exists to show that phthisis, though it occurs at high altitudes, is less frequent there than at lower levels. This fact was noted by Dr. Jourdanet,[9] a French physician living in Mexico, and also by the late Dr. Archibald Smith, who practised at Lima, and it was their observations, including the good effects of the higher regions on those who acquired phthisis in the plains, that led to the development of the Swiss Alpine resorts. Dr. Huggard[10] published the following statistics dealing with the prevalence of tuberculosis among the native population in the Canton Grisons, at different altitudes, in the year 1895:

FIGURES FROM THE CANTON GRISONS, SHOWING DIMINISHED PREVALENCE OF TUBERCULOUS DISEASES WITH INCREASING ELEVATION.

Number of Communes.	Elevation above Sea-level in Metres.	Population.	Average Population of Commune.	Affected with Tuberculosis (480 of the 690 Cases were Examples of Phthisis).	Per Thousand.
(*a*) 15 ...	285- 599	20,369	1358·0	246	12·48
(*b*) 40 ...	600- 999	20,935	523·4	204	9·74
(*c*) 64 ...	1,000-1,499	25,346	396·0	182	7·18
(*d*) 19 ...	1,500-1,880	10,291	541·6	58	5·64

The table indicates, as Dr. Huggard states, "that the prevalence of tubercular disease diminishes steadily with altitude, although the communes of greatest altitude are more populous than the two groups of communes next below them." The figures for the higher regions of Switzerland for the years 1905-1909, published by Dr Schmid,[11] Director of the Swiss Ministry of Health, also show a diminution of the death-rate from phthisis and tuberculosis in general as the altitude increases. It would seem not improbable that the deeper respirations required at these higher elevations, leading to improved blood-supply and better nutrition of the lungs, may explain the diminished incidence of the disease. Dr. William Gordon,[12a] of Exeter, casts doubt upon the influence of mere elevation, and would attribute much of the good effects to the absence of high and rain-bearing winds. These conditions can, however, only be said to hold good during the winter season when the snows have fallen.

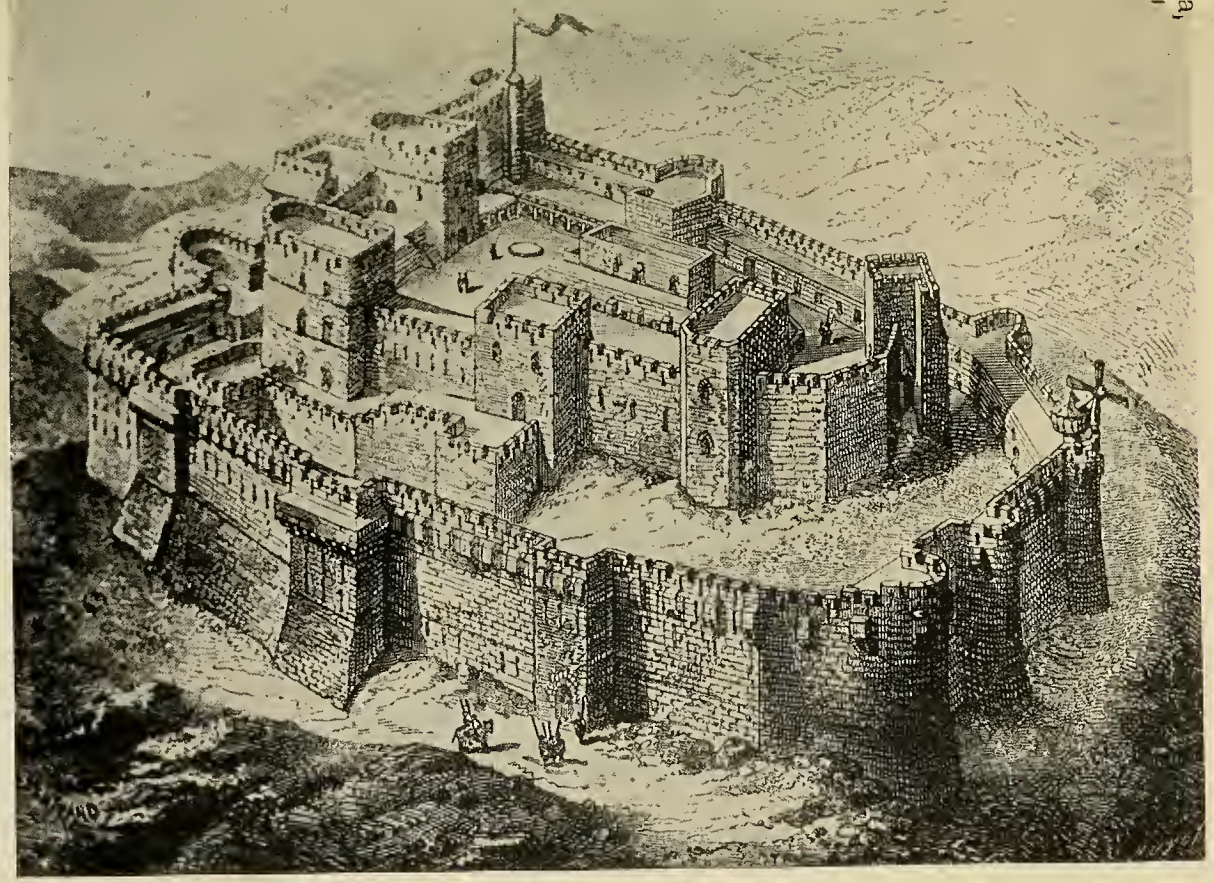
(*b*) *Dampness of the Soil.*—The independent researches of

the late Sir George Buchanan[13] in England, and of the late Dr. H. I. Bowditch[14] in the United States, pointed many years ago to a relationship between phthisis mortality and wetness of soil, the death-rate rising with the dampness of the subsoil. Sir George Buchanan[15] also observed the converse as one of the results of improved surface drainage—namely, that the death-rate from phthisis fell as the drying of the subsoil took place. These results have been criticised, but the observations of Dr. William Gordon,[12d] of Exeter, to which we referred more fully in our last edition, lend some support to the theory. It may be said in brief that towns, villages, hamlets, or houses situated at or near undrained localities on heavy, impermeable soils or on low, level ground, and whose sites, consequently, are kept damp, are more suitable for the development of consumption than those which are placed on dry or rocky ground, or on light porous soils, where the redundant moisture can easily escape.[16]

(*c*) *Exposure to Wind.*—The influence of wind in relation to phthisis was first drawn attention to by the late Dr. Haviland.[17] More recently Dr William Gordon,[12] has devoted much attention to the subject, investigating especially the effect of wind upon the mortality from phthisis in the various districts of Devon. The conclusion at which he arrives is that "populations exposed to strong prevalent rainy winds have a higher death-rate from phthisis than populations sheltered from them." Dr. Brownlee finds this to hold especially with the "young adult type" of phthisis. The matter is difficult to prove, so many other factors having to be weighed and discounted. The ill-effects of winds may be in part direct, but are perhaps more largely due to the closing of doors and windows to which their violence often leads.

3. **Injury.**—Cases of phthisis occasionally come before us in which the symptoms have followed an injury to the chest.

We can recall a remarkable case of the kind, that of a medical man, aged 44, of robust physique, who dated his attack definitely from being crushed against a tree in Scotland by the backing of his motor-car, which he had not sufficiently "braked." Some ribs on both sides were broken, and double hydrothorax followed, with effusion into the pericardium and pneumonia of the left lower lobe. In the blood-stained mucus expectorated a few tubercle bacilli were found. When sent to Sir Douglas Powell by Dr. Burton, of Blackheath, three months later, in December, 1909, there was some shrinking of the left side



Fortress of the Knights Hospitallers, in Syria, taken from the Kurds by the Franks about the year 1125, and rebuilt in 1202. A representation of it as restored. Engraving from *Monuments of the Architecture of the Crusaders in Syria*, by M. G. Rey

Military and Religious Life in the Middle Ages and at the Period of the Renaissance, by Paul Lacroix. Bickers & Son, London

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and dulness over the apex, with a few crackles extending to the fourth rib. He went to a sanatorium, where the sounds became dry, and he put on four pounds in weight. Six months later, although greatly improved, there were still a few bacilli to be found. Dr. Burton sent a final note of this case, stating that, in the summer of 1910, "he had no further trouble with his lungs, and was able to shoot, and could walk most of us off our legs." In 1912 he was seized with acute appendicitis, and died of peritonitis after operation. Dr. Burton knew him well. His health had always been good up to the time of his accident. His mother died at an advanced age; his father early in life, but the cause of death was unknown. He had three children, one of whom died of tuberculosis, aged nineteen.

The subject of "traumatic tuberculosis" has been recently discussed by Dr. Parkes Weber,[18] and it is probable that the cases are to be explained by the injury lighting into activity a latent focus of disease.

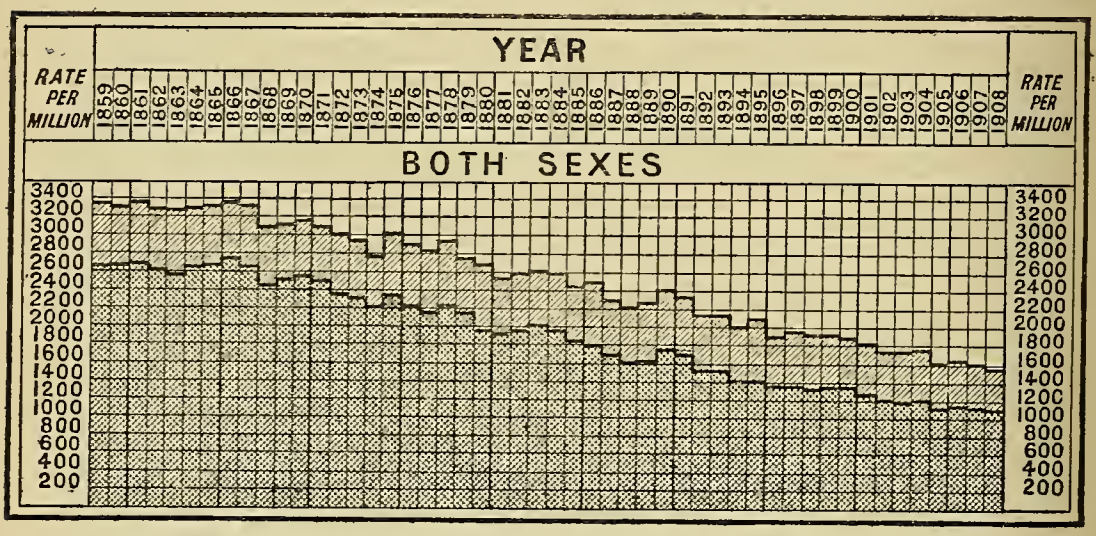
4. **Social Conditions.**—Amongst the general predisposing causes of tuberculosis may be enumerated insanitary conditions of life; debility ensuing upon acute illnesses; debility attendant upon chronic diseases, such as alcoholism, syphilis, anæmia, and gastric affections; hereditary predisposition and mental unsoundness. With regard to most of these factors there is no need for any special remark. It is fully admitted, and in accord with general experience, that those affected with chronic disease and those who are weakly from incomplete recovery from acute illness are prone to be attacked by tuberculosis; that is to say, their resistance to tuberculous infection is for the time, or permanently, lessened or broken down.

An examination of both climatic and industrial conditions predisposing to pulmonary tuberculosis cannot fail to convince anyone how largely they are connected with conditions that fall under the heading of "sociology," as, indeed, we have already indicated when discussing the question of contagion. Phthisis is essentially a scourge of what we call civilisation. Its rarity among nomadic tribes and aboriginal races in all climates, its prevalence in industrial, as compared with agricultural localities, point to social rather than to climatic influences as predominant in the ætiology of the disease. All the depressing conditions of life—anxiety, mental strain, disappointments, poverty, bad sanitation, overcrowding, alcoholic excess, debauchery—are concentrated at the centres of civilisation. The weakly

are helped to live; scrofula, syphilis, rickets, and catarrhs, prevail; the general tone of health is depressed; recovery from acute specific diseases, such as measles, whooping-cough, influenza, and from inflammatory chest diseases, is less complete, and germs, putrefactive and other, are so rife that special precautions against them are necessary to secure the healing of wounds. All that we know about phthisis would lead us to expect its greater prevalence under these conditions, and such we find to be the case.

It is comforting, however, to find from statistical evidence that, with improved sanitation and a general increase in the

Diagram showing Death-Rate per Million living from (*a*) All Forms of Tuberculosis, (*b*) Phthisis; England and Wales, 1859-1908, corrected for Variations of Sex and Age Constitution.



Note.—The darker shading refers to phthisis. (From the Registrar-General's Annual Report.[19])

wage-earning capacity of the labouring classes, there has been a marked and up to recently a continuous decline in the death-rate from this disease. This is well brought out in the above diagram, taken from the Seventy-first Annual Report of the Registrar-General,[19] whereby it will be seen that the standardised death-rate from phthisis, and also from all forms of tuberculosis, has been steadily decreasing. Sixty years ago, it will be noted, the annual death-rate from consumption was 26 per 10,000 living, between two and three out of every 1,000 people dying annually of the disease. In 1908 it was only 11·15, barely more than one person in every 1,000 falling a victim.

Through the courtesy of the Registrar-General we are enabled to give the corresponding figures for succeeding years, thus bringing the table up to date. The standardised death-rates for phthisis per 10,000 persons living are as follows: 1909, 10·63; 1910, 9·88; 1911, 10·31; 1912, 9·91; 1913, 9·60; 1914, 9·93. It must be noted that there has been some check in the rate of decline since 1895, and since 1914 a positive rise, due mainly, though perhaps not wholly, to war conditions, the following being the death-rates for the war years: 1915, 11·53; 1916, 12·30; 1917, 13·83; 1918, 15·20. These figures, for reasons given in the Registrar-General's Report for 1917, exaggerate the increase in the mortality from tuberculosis which has undoubtedly occurred, both among males and females. We shall again refer to this subject.

5. **Age.**—Pulmonary tuberculosis may occur at any age, its heaviest incidence being between the ages of twenty-five and forty-five. The late Dr. Bulstrode,[20] in his valuable report on "Sanatoria for Consumption," pointed out the important fact that the age of maximum mortality from phthisis has become postponed, and now occurs between the ages of forty-five to fifty-five in males, and thirty-five to forty-five in females, instead of between twenty and thirty-five, as was the case in the middle of the last century. This is well shown in the diagrams which we have reproduced (pp. 436, 437) from Dr. Brownlee's[21] recent paper, which demonstrate also that the fall which occurred during the latter half of the last century has been most marked in the young adult period of life. Dr. Brownlee finds that the age-period of phthisis is not constant, as generally supposed, for different parts of the country, being, for instance, much earlier in the Shetland Islands than in London, and he draws the conclusion that it is "not one disease due to one organism, but a group of diseases due to a group of organisms, just as enteric fever and dysentery have proved to be." He speaks of three types or varieties, the "*young adult type*," the "*middle-age type*," and the "*old-age type*," in which the commonest ages of death are from 20 to 25, 45 to 55, and 55 to 65, respectively. The hypothesis is supported by the fact that the two former types at least have a markedly different geographical distribution within the British Isles. An examination of Dr. Brownlee's interesting diagrams suggests the possibility that the varia-

tions of the phthisis mortality in certain districts are due to occupational causes rather than to some specific difference in the tubercle bacilli. Nor must it be forgotten that the incidence of the disease must be reckoned on an average as occurring some years before its mortality, and that with improved methods of treatment this interval between incidence and mortality is steadily lengthening. This postponement of the age of maximum mortality is, however explained, a fact of great importance, in that the victim dies at an age when his family is less dependent upon him, and does not so inevitably lead to that privation among the children which so often is responsible for the further spread of the disease. The exceptional conditions of war service under which so many of our young soldiers have lately acquired tuberculosis may temporarily, in some degree, modify the age of maximum mortality.

6. **Overcrowding.**—Taking the proportion of two persons to a room in tenements of not more than five rooms, as the limit of occupational sanitation, and reckoning, all numbers above that proportion in percentages of overcrowding, it has been shown by Sir Shirley Murphy that in London the death-rate from consumption advances directly in ratio to the overcrowding. This is demonstrated in the following table, taken from the Report of the London County Council:[22]

PHTHISIS DEATH-RATES IN RELATION TO OVERCROWDING.

LONDON, 1901-1909.

(*Taking two persons per room in five-room tenements as the basis.*)

Proportion of Overcrowding in Each Group of Sanitary Areas.	Crude Phthisis Death-rate per 1,000 Persons living.	Standard Death-rate.	Factor for Correction for Age and Sex Distribution.	Corrected Death-rate per 1,000 Persons living.	Corrected Death-rate (London, 1,000).
Under 7·5 per cent.	1·034	1·718	1·00991	1·044	709
7·5 to 12·5 ,,	1·320	1·705	1·01761	1·343	912
12·5 ,, 20·0 ,,	1·413	1·771	0·97969	1·385	941
20·0 ,, 27·5 ,,	1·924	1·805	0·96124	1·850	1,256
Over 27·5 ,,	1·953	1·651	1·05090	2·052	1,394
London	1·472	1·735	1·00000	1·472	1,000

From the above figures it is to be seen that in groups of sanitary districts in London with under 7·5 per cent. of overcrowding the corrected death-rate from phthisis was 1·044 per 1,000 people living. As the overcrowding increased, so did the phthisis death-rate, until, with 27·5 per cent. or more of overcrowding, the corrected death-rate reached 2·052.

The importance of overcrowding as a factor in the spread of the disease is also shown by the fact that in Aberdeen,[23] as stated in 1916 by the Medical Officer of Health, fully one-half of the cases of phthisis notified were found to be occupying the same bed with other inmates, and that at least two-thirds were sleeping with others in the same room. A very similar condition would appear to prevail in Bradford.[24]

7. **Dusty Employment.**—The effect of dust inhaled in the pursuit of various employments was carefully investigated many years ago by the late Dr. Greenhow,[25] and his conclusions as to its baleful influence and the excessive mortality from lung diseases and phthisis which results from it, have been borne out by later observers.

This subject we have referred to in the chapter dealing with pneumonokoniosis, and we may recall that it is in the trades in which the dust particles are hard and gritty, such as gold-mining, tin-mining and lead-mining, and among cutlers, potters and grinders, that the danger is greatest. Soft particles, such as coal dust and the animal dusts produced from bone, horn or ivory, are less harmful, and, indeed, among coal-miners the mortality from phthisis is below the average. These facts are illustrated by the researches of Dr. John Tatham,[26] who showed that for the three years 1890 to 1892 the death-rate from phthisis among potters, lead-miners, cutlers and file-makers was between three or four times that of the agricultural labourer, whilst among tin-miners it was nearly five times as great, and these figures are fully maintained in the recent Reports for 1918 and 1920 by Dr. Brownlee. The mortality, in fact, among tin-miners proved higher than that of any other trade examined, the other four mentioned coming next in order of frequency. Dr. Tatham's figures show also in the dusty trades a greatly increased mortality from other diseases of the respiratory organs.

The observations of Dr. Scurfield,[27] Medical Officer of

Health for Sheffield, are also of importance, demonstrating that although the total death-rate from tuberculosis in Sheffield shows nothing peculiar, yet on analysis it is found that the male death-rate is much greater than that for England and Wales, while the female mortality is much smaller. Further examination shows that the excessive mortality from phthisis among the Sheffield males is due to the great prevalence of the disease among the grinders, and to a less extent among the cutlers. The following figures abridged from his table are instructive, showing that amongst the grinders half the mortality is due to consumption, and two-thirds to diseases of the respiratory system, as opposed to one-sixth and one-third respectively among the total male population of the town. The same holds good with the Cornish miners.[28]

MORTALITY AT SHEFFIELD AMONG MALE WORKERS IN CERTAIN TRADES DURING THE SEVEN YEARS (1901-1907).

Occupations.	Numbers.	Mortality (Rate per Thousand living).		
		All Causes.	Phthisis.	Respiratory Diseases.
Grinders	3,868	30·9	15·0	5·6
Cutlers	3,889	30·0	6·0	7·0
Tailors	941	21·4	1·4	4·1
Printers	487	17·3	3·8	2·6
Joiners	2,286	13·9	1·7	2·6
All males (1905-6-7)	—	16·2	2·6	2·1

A consideration of the above facts can leave no doubt as to the danger resulting from the inhalation of irritating dusts, the damaged lung being thereby rendered more easily a prey to invasion by the tubercle bacillus.

8. **Alcohol.**—This is believed by many to be a potent factor in the ætiology of the disease, and French statistics are quoted as showing a parallelism between the death-rate from tuberculosis and the amount of alcohol consumed. In England, according to our experience, the majority of phthisical patients show no evidence or history of alcoholism, and for ourselves we doubt whether alcohol is *per se* a favouring agent to tubercle. There can be no question that, if taken in such excess as to produce the diseases of chronic alcoholism—

FIG. 44

Diagram I.—Showing the Percentages of Phthisis Deaths to Total Deaths of Persons from all Causes, London, 1631-1917. (Dr. Brownlee.)

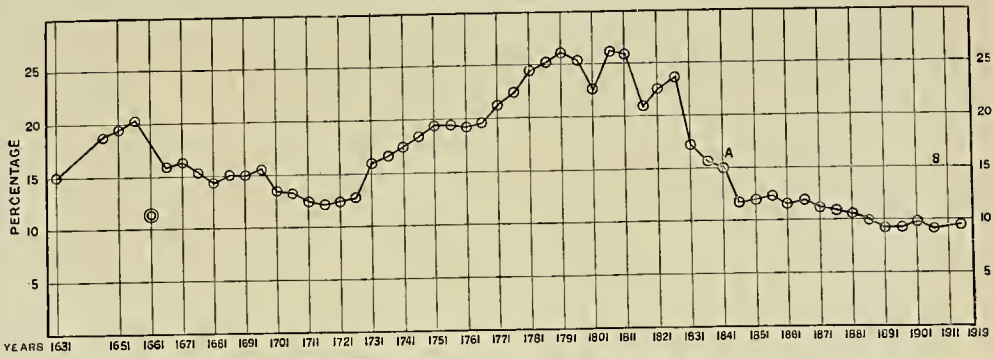


Diagram II.—Showing Percentage of Phthisis Deaths to Total Deaths from all Causes, London, 1851-1919, Subdivided for Males, Females, and Persons. (Dr. Brownlee.)

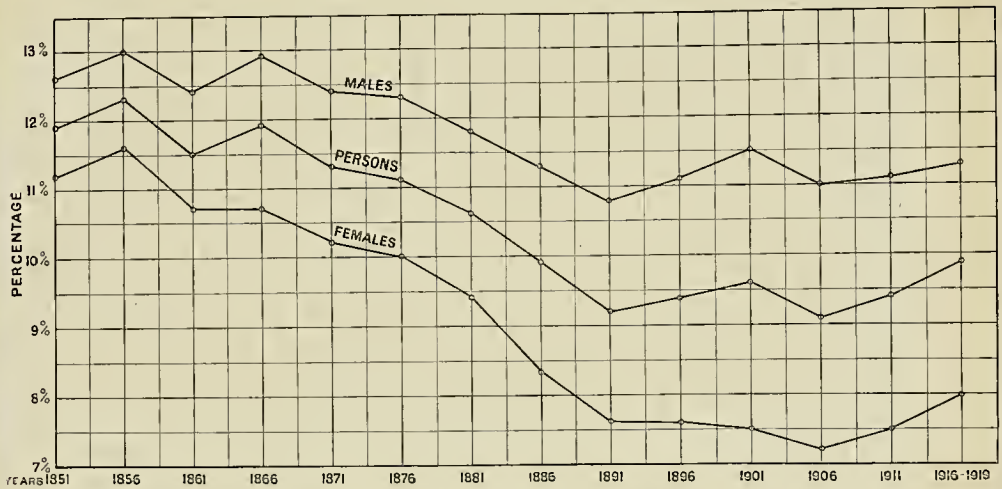
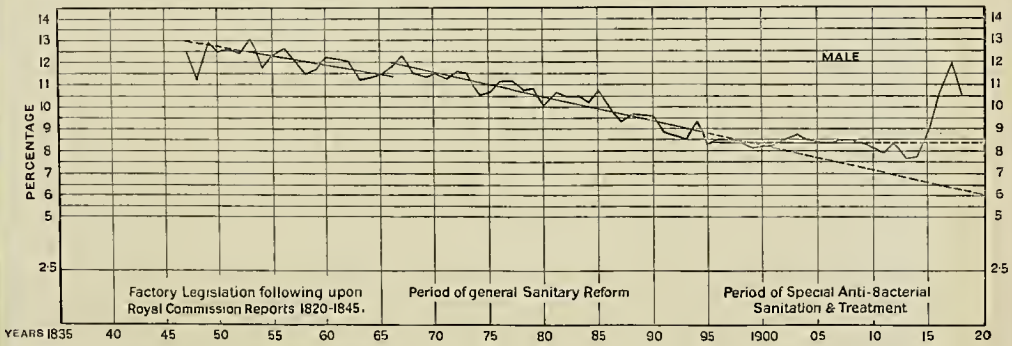


Diagram III.—Showing Deaths from Phthisis as Percentage of Deaths from all Causes, England and Wales. (Prof. Karl Pearson.)



cirrhosis, alcoholic bronchitis, pharyngitis, or peripheral neuritis—it does beget a tendency to the acquisition of tubercle; even then, however, the disease is apt to run a chronic course. A more important way in which chronic alcoholism favours tuberculosis is indirectly by leading people into dirty, ill-ventilated, and contaminated drinking-places, in which the virus of tubercle is diffused.

9. **Insanity.**—Mental unsoundness is, again, in all probability only indirectly operative in the ætiology of phthisis, insane patients being admitted into asylums which have been in the past to a certain extent insanitary and overcrowded, and which are liable, from the habits of such patients, to specific contamination. The yearly average of 1,809 deaths from phthisis in lunatic asylums which obtained in 1912 to 1914 increased to 5,605 in 1918,[29] no doubt attributable to the overcrowded conditions that prevailed during the war.

10. **Epidemic Features.**—We may finally ask whether behind and beyond all the ætiological factors we have been considering there may not be some other influence at work controlling the incidence of tuberculosis. We annex a diagram (Fig. 44) from Dr. Brownlee's Report in 1918 to the Medical Research Committee on the Epidemiology of Phthisis,[21] in which he illustrates the curve of death-rate from the disease in London, which he has traced from the Registrar-General's Reports and such information as is available before they were instituted. Diagram I. depicts a long epidemic wave of phthisis through the whole of the eighteenth century, reaching its height at the commencement of the nineteenth century, when for about thirty years it fluctuated, accounting for between 26 per cent. and 20 per cent. of the total deaths, and then declined to about 10 per cent. at the end of the century. Professor Karl Pearson also, in general agreement with Brownlee, further shows that within the period covered by the Registrar-General's Reports, from 1841 up to 1910, there is evidence of the disease being in some manner governed by influences other than those of mere environment. As will be seen, the curves of mortality for the whole country given in Diagram III. show a moderate declension during the first thirty years in which the Factory Laws were in force; a more decided decline during the second period of twenty-five years, from 1865, when general sanitary reform prevailed; and

then a failure proportionately to respond in the third period of twenty years to the special sanitary and therapeutic measures based upon a recognition of the specific cause of the disease. This latter check in the declension of the death-rate, it will be observed, began before the war, and although increased to a positive rise in death-rate during the war, is not wholly attributable to that cause. The general death-rate, on the other hand, remained constant through the first period of factory laws, and then fell steadily and without check through the second and third periods; in the last period, indeed, the decline being still more marked. When we come to analyse these somewhat discouraging results in the light of the detailed statistical researches related in Dr. Brownlee's two reports and in Professor Karl Pearson's records, to which we have extensively alluded, some very instructive facts are disclosed, and some interesting speculations are raised relating to the prevalence and biology of tubercle.

We have already referred to the three types into which Dr. Brownlee believes phthisis to be separable. He finds that the main decline for the last sixty years, up to 1910, has been due to a diminished mortality—from 1·52 to 0·24 per cent. for males, or nearly 7 to 1—amongst the lives of the *young adult type* (age twenty to twenty-five); the middle-age type, of the period between thirty-five and fifty-five, maintaining practically the same mortality during that time—1·12 to 1·16 per cent. The decline in mortality amongst the younger lives has been most marked in towns; in country districts and exposed situations the disease amongst them being proportionally more prevalent. Wind and weather and subsoil take an important part in its ætiology, the interesting diagrams in Dr. Brownlee's first report (1918) showing its prevalence in our wind-stricken districts of North Scotland, Wales, and Devonshire.[21a] It is, on the other hand, but little affected by insanitary environment.

Amongst the lives of the *middle-age type* the decline in death-rate has been much less manifest, and has been in closer conformity with the general death-rate. This type prevails especially in urban districts, and a glance at Dr. Brownlee's diagrams will show a much closer association with industrial conditions. All the occupational causes of phthisis are mainly operative towards the middle periods of life. And it is

in accord with experience that hereditary influence is a more important factor in the young adult period, whilst in the middle-age period the disease is more definitely acquired, from prolonged exposure to adverse environment, combined with other exigencies in the struggle and harassment of middle life.

There are some considerations that militate against any dogmatic acceptance of the first two types of phthisis as having been fully established. Dr. Brownlee suggests them without dogmatism. In his statistics, which are of death-rates only, some qualification is perhaps needed from the fact that many young people afflicted with chest delicacy or actual disease in urban districts are sent away to the country, whilst many cases "recover" under treatment, and with a recurrence in later years such cases tend to load the middle-age group.

It would appear, from a due consideration of all the circumstances of ætiology, that the tubercle bacillus is sufficiently ubiquitous to render all persons liable to attack; and, indeed, most persons under civilized conditions of life are at some time or other attacked; but that under conditions of concentration of the poison in overcrowded localities and in dirty and dust-laden environments the contagion may be so strong as to be operative against the resistance of those who have no original constitutional liability to the disease. The hereditary factor tends, on the one hand, to increased vulnerability to early attack, and, on the other, to gradual increase of immunity of the race by extinction of the more susceptible and survival of the more resistant.

The biology of the tubercle bacillus has yet to be traced to its probable saprophytic ancestry, and the morphology of its parasitic phases to be further disclosed, before we shall be in a position fully to understand its various types and degrees of virulence, the reactions of its host, the animal organism, with regard to it, and the abiding source of its continuance through the ages. As remarked by the Research Committee in their introduction to Dr. Brownlee's first report of 1918, the decline in mortality in past decades "is most naturally to be regarded as the ebb of a long epidemic wave, to be succeeded perhaps—indeed, probably—by the rise of the next wave in its turn, unless science can find the way of interference, and its explanation can only be given by further study."

Our primary efforts—perhaps better directed—to maintain its decadence must not be relaxed; but the true biology of the disease has yet to be found.

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CHAPTER XXXI

PULMONARY TUBERCULOSIS—(*Continued*)

Pathology.

On inspecting the lungs of one who has died of phthisis, we meet with a great variety of appearances, which may, nevertheless, be recognised as the results of a comparatively few morbid processes. We see consolidation of the lung in every stage of formation, decay, and removal; and, glancing at the emaciated form before us, we have a practical definition of phthisis pulmonalis or consumption—viz., progressive consolidation and decay of the lung with progressive wasting of the body.

This destruction of lung and waste of body are associated with two striking morbid processes in the lung: (1) the occurrence of the characteristic granulation of tubercle, disseminated through the organ, or collected into nodular groups, or mingled with inflammatory changes, developing into fibroid tissue, or immediately undergoing necrotic change; (2) the appearance of areas of inflammation, generally also tuberculous in origin, and affecting with varying degrees of intensity the different tissues of the lung. Such inflammatory processes run an acute, chronic, or chequered course, and usually terminate in caseation and softening.

In addition to the above, two further processes will require our attention in considering the pathology of the disease, viz.: (3) the penetration of the lungs by secondary organisms; (4) the pathological changes in the lungs and pleuræ which result mechanically from the respiratory movements of the chest wall. Let us now consider each in turn.

(1) *The Tuberculous Nodule.*—The tubercle bacillus, as we have seen in the preceding chapter, is brought to the lung either by aspiration into the fine bronchi or alveoli, or, more rarely, by means of the blood-stream, from some more or less

distant part of the body. Whenever it finds a suitable resting-place and the conditions are favourable, it commences to develope, and to cause a proliferation from the fixed cells of the part, whether bronchi, alveoli, or capillaries, resulting in a collection of epithelioid cells. Later, in the centre of the little mass, one or more giant cells may appear, and on the exterior a zone of leucocytes, the whole thus constituting the perfect microscopical tubercle. The presence of giant cells and leucocytes will depend, however, to a great extent upon the activity of the disease. If virulent, giant cells will be but rarely seen; if less active, they will be more evident, and leucocytes proportionately fewer. Giant cells are thus not an essential part of tubercle, as was once imagined; and, we may add, they are not restricted to it, being found in many other morbid products. If suitably stained, one or more tubercle bacilli will probably be detected in the nodule, sometimes within the giant cells, but in the human subject more commonly between the cells. No vessels are present in the tubercles, although a vascular zone often surrounds them. From their close contiguity to vessels, however, their nutrition is assured, until the toxines excreted by the contained bacilli cause their degeneration and caseation.

Wherever started, the tuberculous process tends quickly to invade adjoining alveoli, setting up a tuberculous broncho-pneumonia, which is very apt to caseate, the patches appearing to the naked eye as minute yellow tubercles. The disease extends by aspiration to alveoli as yet unaffected, or through the lymphatics to other portions of the lung. In acute miliary tuberculosis, when life is rarely prolonged beyond a few weeks, the tubercles at the time of death are transparent, or only just commencing to caseate. In the more chronic forms of the disease, however, we often meet with a somewhat larger and firmer variety of granulation, hard, semi-transparent, horny-looking, and of about the size of a pin's head, intimately connected with the surrounding tissue. This fibroid transformation of the tubercles occurs especially in those which have their origin in lymphatic vessels or in the interstitial connective tissue of the lung, the alveoli being but little involved.

In order to apprehend how the disease thus extends and is disseminated, we must recall to mind that the whole lung is

pervaded in every crevice of its structure by lymphatic tissue, consisting of branched protoplasmic cells communicating with fine tubes and interstitial spaces lined by endothelial cells. The late Sir John Burdon Sanderson observed the sheaths of the minute bronchi as being favourite sites for the origin of such tuberculous granulations; other common sites are the lymphatics of the alveolar walls, and of the perivascular and subpleural tissues.

We may here observe that there are two varieties of degenerative change to which tuberculous processes are liable, and that whilst *caseous necrosis* in different degrees of intensity is the process that especially awaits the inflammatory form of tuberculosis of the lung, *fibroid change* is the more characteristic sequel to the neoplastic development represented by the firm, semi-transparent granulation which we have been describing. The peculiar form of fibroid tissue into which, under favourable circumstances, tubercle thus becomes converted is at first easily recognisable from ordinary hyperplastic fibrous tissue. Subsequently it gives rise to bands or tracts of uniform homogeneous texture. Finally it may degenerate and be removed, leaving merely a pigmented point and a more or less tiny stellate wrinkle. This conversion of tubercle before its final decay has, perhaps, hardly been sufficiently insisted upon as an essential character always observable, if circumstances permit the attainment of the necessary stage. It is of importance as affecting the clinical characters of chronic tuberculosis. In acute tuberculosis the patient does not often live long enough for any process of the kind to take place. In the chronic form of the disease, however, and when tubercle attacks a lung rendered quiescent by previous consolidation, the stages of development of tubercle into fibroid tissue may be seen.

(2) *Tuberculous Pneumonia.*—There is a great difference, both histologically and clinically, between the purer form of tuberculous lesion, of which we have been hitherto speaking, and those cases in which the organism produces chiefly an inflammatory process in the pulmonary tissue. In its gravest form this leads to a pneumonic infiltration of the lung, in coalescing centres, inevitably proceeding to caseous necrosis of the parts affected, a condition which will be described in a succeeding chapter under the heading Acute Pneumonic

Phthisis. It was long ago pointed out by Addison[1] that the inflammatory process takes the largest and most important share in the production of the various appearances met with in pulmonary tuberculosis, in which disease it is the destroying element.

With regard to the caseous necrosis, it may be stated that, although the fatty change which is so important an element in the process is common to many inflammations, and a chief means by which their products become eliminated or absorbed, yet caseation is a more uniform result of tuberculous inflammation than of any other, and it is almost synonymous in the lung with the "crude or yellow tubercle" of older writers. The distinguishing feature of tuberculous caseation is the presence therein of the tubercle bacillus.

With true lobar pneumonia we have but little to do in dealing with cases of phthisis; we only meet with it as an exceptional complication. The form of pulmonary inflammation which is most commonly met with is of a different kind, and, except in the very acute forms already referred to, it is more insidious in onset. In form it is broncho-pneumonic, involving the minute bronchi and infundibula of the lung over areas of variable size around each centre of infection. The coalescence, however, of many adjacent foci may sometimes cause the consolidation of a whole lobe. Such inflammation may be due to the tubercle bacillus alone, but the action of the bacillus is assisted in certain cases by other organisms, and to this matter we shall again refer.

Most commonly the cellular products of this catarrhal inflammation, after having undergone more or less complete caseous necrosis, break down and are eliminated. Less frequently they become inspissated by absorption of fluid matter, and remain for a long time—perhaps for the lifetime of the patient—in a cheesy condition, quiescent, but a source of possible danger in that they contain in many cases living tubercle bacilli, the activity of which may be reawakened should the patient's general vitality become lowered or the local resistance in the lung be diminished by intercurrent catarrhs and congestions. In other cases calcification ensues, but, even in such cases, the patient is by no means always free from further risk.[2]

In these inflammatory processes the elastic tissue of the

lung plays no active part. It escapes but little altered when the alveoli break down, and, when recognised in the sputum, affords certain evidence of pulmonary destruction.

In addition to the parenchyma proper of the lung, which, with its epithelium, is the special seat of catarrhal pneumonia, there exists the fibrous stroma, if we may so style it, formed by the interlobular areolar tissue, supplying sheaths to the vessels and bronchi, contributing also to the formation of the alveoli, and intimately connected at the surface of the lung with the investing pleura. It could not be expected that an inflammation of the lung of any great severity would leave this widely spread tissue unscathed; and it might also be anticipated that a tissue thus, comparatively speaking, deeply placed would, as a general rule, only be affected secondarily to disease of the parenchyma or pleura. And this we find to be the case. From this interstitial tissue, under conditions of inflammatory irritation, are derived, in great part, the tough, fibrous, pus-secreting walls of cavities, and the trabeculæ, which for a long time resist the most severely destructive processes. The inflammatory process in this tissue is as a rule a more deliberate one; even when in a state of active ulceration, as in the walls of certain cavities, the destruction is molecular; sphacelus is rare. The inflammatory reaction, indeed, more generally partakes of the character of growth under irritation, producing *fibrosis* of the lung, and sometimes this condition may become so extensive as to constitute the predominant disease. To such cases the name "fibroid phthisis" has been applied. Decay, however, in most instances, finally sets in; the fibrous tissue, at first merely hyperplastic, loses its character as such; its nuclei, at first abundant, gradually fade; its fibres fuse into tough homogeneous bands, and in their turn become granular and fatty, and finally crumble away.

(3) *Mixed and Secondary Infections.*—We have already hinted that, although the tubercle bacillus plays the specific and predominant rôle in the lesions of phthisis, there are other organisms to be reckoned with in its pathology, among which the streptococcus, staphylococcus aureus and albus and the pneumococcus would appear the more important. We must now consider what part these play in the evolution of the disease. That such organisms are often present in the sputum of phthisical patients, being responsible for some

measure of the bronchial catarrh, which is so common a feature of the disease, and which may be regarded in part as due to secondary infection, is a fact which has long been known, but it is a question much debated how far they play an *active* rôle in the spread of the tuberculous disease. Certain observers urge that, as fever, wasting, caseation and softening can all be produced directly by the tubercle bacillus, these manifestations of the disease should be attributed to the bacillus, and to it alone. The work of Sorgo,[3] both clinical and pathological, would, however, appear to show that even in sanatorium patients, in whose sputum under the purer atmospheric conditions the bacterial flora becomes markedly diminished, true mixed infections, in which the tuberculous granulations are the seat both of tubercle bacilli and other infecting organisms, do, though rarely, occur. This has been confirmed at the King Edward VII. Sanatorium by Dr. J. A. D. Radcliffe,[3] to whose Weber-Parkes Prize Essay we must refer the reader for full technical details, and for a critical consideration of the literature of this important subject. Dr. Radcliffe, following Sorgo's method, treated the sputum by repeated washings, and in this way was able to separate from the tubercle bacillus, by mechanical means, organisms not derived from the actual focus of disease. He examined in this way the sputum of thirty patients suffering from febrile phthisis, and in twenty-five he obtained a pure growth of the tubercle bacillus. In the remaining five other organisms grew as well, showing that they were intimately associated with the tubercle bacillus in its activity, and these cases we may regard as true examples of mixed infection.

We may conclude, therefore, that even under sanatorium conditions such infections do occur, but only in a small proportion of cases (though in Dr. Radcliffe's experience those of graver outlook), and that the activity of the disease and its febrile manifestations are in most instances the result of the unaided action of the tubercle bacillus. That under less favourable conditions mixed and secondary infections may be of more frequent occurrence is possible and even likely, and this view is supported by the improved results obtained by sanatorium treatment, under the purer air conditions of which the opportunities of such infection are diminished; also by the rapid spread of the tuberculous disease, which sometimes results when influenza attacks a phthisical patient.

(4) *The Mechanical Effects of the Rigid Chest Wall and Respiratory Movements.*—The respiratory movements of the chest cavity take part in the pathology of phthisis, as of other thoracic affections, but in a more marked degree.

When the lung disease is of a chonic, indurative, contractile character, the effect of the continued thoracic efforts to expand the toughened lung is to stretch any adhesions which may have formed, and to separate the pleural layers; the further contraction of the lung continues the process, so that the parietal and visceral pleuræ may become separated by an interval of as much as half or three-quarters of an inch. This space is at first filled by serous fluid effused into the loose meshes of the areolar tissue of the stretched adhesions. In this way is produced the œdematous pleura (Plate XXVIII., *Frontispiece*). At a subsequent stage of the disease, by the continued growth of the areolar tissue, the whole space becomes occupied by tough fibrous material, and the two layers become welded together into one uniform layer. That this is the real history of the enormous thickening of the pleura in many cases of chronic pulmonary disease we have satisfied ourselves by repeated observation.[4] Thickening of the pleura has in the past been regarded too much in the light of a dangerous pathological process, liable to extend into, and by its contractile power to compress, the proper lung tissue. It is generally, in fact, a condition secondary and quite subsidiary to the lung disease.

In primary pleuritis the thickened pleura is produced in a different way. After absorption of the fluid, a certain thickness of lymph remains between the two layers of the pleura, into which granulations from each surface penetrate, and finally unite, completing the adhesion.

There are, however, not a few cases of phthisis of the pneumonic kind of tolerably acute progress, and attended with little contraction, in which, though the pleural surfaces are inflamed and covered with finely granular lymph, they do not become united. It is in these cases that pneumothorax is especially likely to occur.

The constant movement of the lungs no doubt goes far to modify and hasten the progress of morbid processes going on within them, and the constant access of air to the diseased surfaces tends to maintain suppuration and to promote the

absorption of septic matters. By the inhalation of portions of sputa, laden with acrid and specific poison, to distant parts of the lungs, the lesions of phthisis are further disseminated.

Site and Spread of the Lesions.—In pulmonary tuberculosis of the adult the disease usually manifests itself by a deposit of tubercle near the apex, perhaps more often of the left lung. From this primary focus it extends downwards through the lung, though before it has proceeded far the opposite apex becomes, as a rule, affected. The march of the disease from apex to base follows a more or less definite progress, seen best in chronic phthisis, and which has been carefully described by Sir James Kingston Fowler.[5] Our own somewhat extensive post-mortem experience leads us substantially to accept the correctness of his conclusions.

The first lesion is situated, not at the extreme apex of the lung, but about an inch below its summit. From this point, possibly as the result of inhalation under the influence of gravity in the supine position, the disease spreads backwards, so that physical signs soon manifest themselves in the supraspinous fossa. Scattered tubercles next make their appearance in the front of the upper lobe, gradually extending downwards from the apical focus. Before, however, such extension has proceeded far, lesions manifest themselves at the apex of the lower lobe of the same side, and from this point they extend, by means of scattered areas of infiltration, often of the nature of racemose tubercle, towards the base. As, however, Sir James Kingston Fowler pointed out, a special zone of infiltration often shows itself in the lower lobe along the line of the great fissure, so that moist sounds are often audible in this region as far as the anterior limit of the fissure.

Before the disease has extended far in the lung first attacked the opposite apex becomes in most cases affected, whence the disease progresses in a manner very similar to that already described in the other lung. The middle lobe is, in our experience, affected only late in the disease, and rarely to a considerable extent.

Occasionally physical signs are most marked at the base of the lung, and if they affect the whole lower lobe and tubercle bacilli are present in the sputum, they may be accepted as indicating tuberculous disease, the case being one of so-called *basal phthisis*. In such cases, which are only of rare oc-

currence, signs of an old arrested lesion at the apex will not uncommonly be found.

Hilum Tuberculosis.—Recently another variety of phthisis has been described, the so-called *Hilum tuberculosis*, or, as it is termed by radiologists, *Peribronchial phthisis*, in which the disease is thought to commence in the bronchial glands and thence to spread fanwise through the lung along the course of the bronchi and vessels.

In children the bronchial glands are not infrequently infected with tubercle, and may become caseous, a condition which, as Professor Ghon[6] and Dr. Canti[7] have shown, is in most cases secondary to a primary focus in the lung. The attack may pass and the lesion subside into obsolescence, and may so remain for a long time, even for life. In other and less common cases the disease in the glands extends, spreads beyond the capsule, and directly invades the lung. Such cases of pulmonary tuberculosis have long been recognised in children.

In the adult we believe that cases of pulmonary tuberculosis originating in the glands, if they occur, are very rare. The numerous autopsies which we have ourselves performed in cases of tuberculosis have not led us to doubt the accuracy of the older view, that in phthisis the disease in the great majority of cases commences near the apex, and thence, as we have already indicated, gradually spreads downwards through the lung. It is also interesting to note that in phthisis in the adult the bronchial glands are not, as a rule, caseous. In 263 cases in which we investigated this point,[8] we found the glands enlarged in 48·2 per cent., and showing areas of caseation in only 9·1 per cent. In a further 14·8 per cent. old calcareous foci were present. The condition, therefore, of caseation, in which extension into the lung might most reasonably be expected, is not of frequent occurrence. The recognition of hilum tuberculosis is, in fact, largely based upon the occurrence of shadows seen in screen examination and X-ray plates, which radiate into the lung from the neighbourhood of the bronchial glands, which themselves often in the adult show evidence of old disease. Such radiating shadows, in the absence of post-mortem control, are notoriously difficult to interpret, and may have more than one explanation.

The mechanism of extension of the infection from the hilum

through the substance of the lung to its periphery is also somewhat difficult to recognise. The supposed extension of the infection is against the current of the lymph stream, though we are aware that this is not an insuperable objection. Arterial conveyance of the infection more usually assumes the form of acute general miliary tuberculosis. Dr. Riviere,[9] who strongly advocates hilum tuberculosis as a separate variety of the disease, would account for the antecedent pleurisies of phthisis by extension from the hilum through lung to pleura; but we conceive their origin to be, in the particular cases referred to, more frequently by direct infection of the pleural surface through the serous fluid.

Our view, then, with regard to hilum tuberculosis is that it is an unnecessary addition to nomenclature, and we think that in the adult there is reason to doubt the soundness of the view that tuberculosis spreads continuously outwards from foci at the hilum. With other observers, we have met with cases in which, in the absence of obvious lung disease, the glands at the hilum have been the seat of active tuberculous infection. Such cases have manifested themselves by continued pyrexia, and on X-ray examination the hilum glands have been seen to be considerably enlarged and, no doubt, diseased. This variety constitutes, in fact, a true hilum tuberculosis, but it is not this form of disease to which the term is usually applied.

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CHAPTER XXXII

ON THE VARIETIES OF PULMONARY TUBERCULOSIS. ACUTE TUBERCULOSIS OF THE LUNGS

LAENNEC maintained phthisis to be a specific disease, and the result of Koch's discovery of the tubercle bacillus as the virus in all cases has been to reinstate his view. It cannot, however, be questioned that the range of pathological diversity within the sphere of this single malady is so great as to justify its subdivision, for clinical purposes of prognosis and treatment, into subgroups and varieties. Without further apology, then, we will endeavour to sketch the main features of the following types of the disease:

Acute tuberculosis of the lungs—

(1) Confluent form ... Acute pneumonic phthisis; *synonyms:* caseous pneumonia, lobar form of caseous tuberculosis of the lung.

(2) Disseminated form
- Florid phthisis; *synonyms: phthisie galopante*, broncho-pneumonic caseous tuberculosis.
- Acute miliary tuberculosis.

Subacute tuberculosis of the lungs—

Pulmonary tuberculisation.

Chronic tuberculosis of the lungs—

Chronic pulmonary tuberculosis, ordinary chronic phthisis.

Fibroid phthisis.

There are further to be considered separately, on the ground of mere convenience, certain of the prominent con-

sequences of phthisis, about which are grouped some of the most characteristic features of the disease, viz.:

Tuberculous excavation of the lung—the cavity stage of phthisis.
Hæmoptysis.
Laryngeal tuberculosis.

Pneumothorax, a sequel of more rare occurrence, has been already fully dealt with in a preceding chapter.

Acute Tuberculosis of the Lungs.

I. Confluent Form.

Acute pneumonic phthisis. *Synonyms:* **Caseous pneumonia. Lobar form of caseous tuberculosis.**

In cases of this type, the pathology of which we have described in the preceding chapter, the consolidation is extensive and dense; the disease is more "massed" in the affected lobe than is the case in the more chronic varieties, and occupies, perhaps, its entire extent. As a rule the initial attack is one-sided, and the apex of the lung is the part affected, but in more than one case we have known the base first attacked.

This form of the disease is apt to be confounded with croupous pneumonia, the signs of consolidation being uniform over an extended area. Bronchial breath-sounds are heard, and crepitations, at first fine, then coarser, with bronchophonic voice-sounds. The onset is abrupt and stormy, with pain in the chest, short cough, scanty expectoration, sometimes attended with hæmoptysis, a raised temperature, a rapid pulse, and quickened breathing.

Many of these massed consolidations of the lung, indeed, consist in part of areas of simple pneumonia, and it is only after the resolution of these portions that the tuberculous nature of the case is clinically unmasked with the softening of those centres which have undergone caseation.

In the distinction of these cases from the not uncommon instances of true pneumonia of the apex, the following considerations are important:

(1) The temperature, although maintained above the normal, has even from the first a more fluctuating range than that of

pneumonia. The tongue is as a rule moister and less coated, and hectic sweatings soon become marked.

(2) No critical fall of temperature corresponds with, or follows upon, the appearance of moist sounds.

(3) The hæmoptysis, if present, is decided, not a mere rusty colouration of the phlegm. The sputum is also more abundant, and from the first more or less purulent, and soon comes to contain bacilli and alveolar elastic tissue (Fig. 45).

(4) The leucocyte count in the blood is stated by Drs. Gulland and Goodall[1] to be usually below normal; in pneumonia, on the contrary, a leucocytosis is, as we have seen, commonly present.

FIG. 45.—ELASTIC TISSUE AND FRAGMENT OF SMALL VESSEL, FROM EXPECTORATION OF PATIENT WITH RAPIDLY FORMING CAVITIES (DRAWN BY DR. SYDNEY COUPLAND). × 200.

(5) As the case proceeds, amid the moist crepitant râles, which cannot be distinguished from those of resolving pneumonia, larger clicks are heard in several centres, which become more liquid and characteristic of pulmonary softening.

(6) The breath-sound, which had become more or less completely masked by the numerous râles, again appears in patches corresponding with the large clicking râles, and assumes a hollow tubular quality. The voice-sound over these areas becomes of a nasal snuffling quality, and finally there is distinct pectoriloquy.

(7) The rapid emaciation and markedly hectic temperature of the patient, his profuse sweatings, and the increasingly

copious and purulent expectoration, within a few weeks put aside all possible error of diagnosis. Long before this, however, the true nature of the case will have been suspected, and the diagnosis established by the finding of tubercle bacilli after one or more examinations of the sputum.

Cases of this form of tuberculosis by no means all exhibit at the first onset the stormy symptoms above sketched. But in all the physical signs rapidly extend, until a more or less considerable portion of one lung is involved, before the signs of breaking down into cavities are manifested.

In persons of "strumous" type, who are common subjects of this variety of phthisis, the fever may not be sufficiently violent, nor the other symptoms urgent enough to prostrate them in bed; but remittent fever, cough, expectoration and hectic sweatings are invariably present, and sufficiently manifest to excite alarm in parents and friends.

It is remarkable, however, to observe the rounded contour of features and the relative plumpness of figure maintained by some patients even far on in this form of the malady, which is certainly most common in young girls and women. The features are usually pale with anæmic mucous membranes, but in the younger subjects the cheeks may be well coloured. The chest is usually of fairly good formation, and, in common with the rest of the frame, well covered with a layer of adipose tissue. The muscular system is, however, poorly developed and languidly innervated, the pulse feeble, and the nervous system wanting in tone.

In the form of tuberculosis now under consideration there is, in spite of its initial severity, not infrequently a tendency to arrest. This is brought about by: (1) elimination of the caseous products; (2) cicatricial contraction of the cavities thus formed; (3) compensatory development of the opposite lung.

It is a mistake commonly and naturally enough made by the inexperienced observer, on having revealed to him through his stethoscope the signs of breaking down of pulmonary consolidation in one or more centres, to infer that a further step has been advanced towards a hopeless prognosis in the case before him. In a sense, and while fully admitting the gravity of the case at the first moment of diagnosis, these phenomena of softening are equally essential and important

steps towards amendment. Without them it is impossible for healing changes to ensue.

At the same time, this process of elimination is attended with dangers of its own which must be reckoned with.

(*a*) Suppurative fever necessarily attends the process. A fluctuating hectic temperature, night sweats, cough, and more or less profuse expectoration, containing elastic tissue and bacilli, are present. Hæmoptysis rarely occurs to any great extent. The character of the temperature chart marking this period of phthisis (Fig. 46) is precisely the same as that of any imperfectly drained abscess, wherever situated.

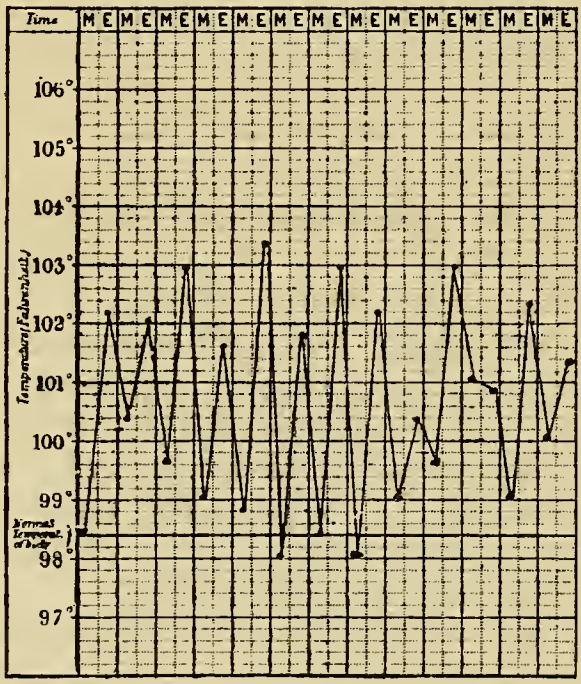

FIG. 46.—ANNIE T.: FLUCTUATING HECTIC TEMPERATURE; ACUTE PNEUMONIC PHTHISIS; CAVITIES RAPIDLY FORMING.

(*b*) There is throughout this period a danger of the opposite lung becoming involved in separate centres, from inhalation of specific morbid products in course of expectoration. The possibility of secondary miliary tuberculosis at this stage is also not to be forgotten, but it is, so far as we have observed, a complication of much more rare occurrence than in other stages.

(*c*) The patient tends to become more and more exhausted, and to lose weight throughout this period, and

in severe cases constitutional resources are seriously taxed by a downward course of from six weeks to three or four months.

During the process of elimination cavities gradually become manifest to auscultation, and perhaps increase by coalescence to form one or two amphoric areas. Some shrinking of lung from loss of substance may be observed, the heart's surface being uncovered more or less to right or left, according to the side affected.

Coincidently, however, with these alarming signs of lung destruction, in a goodly number of cases more hopeful features may be observed, which point towards the arrest of the disease. Thus:

(*a*) We may note that the opposite lung has not become affected to any material extent, and that the limits of the disease on the affected side have not extended, but have perhaps even retrenched. It is necessary here to warn the auscultator against mistaking morbid sounds heard on the healthy side, but really consonated from the affected side, for signs of new disease.

(*b*) Before the subsidence of hectic phenomena, careful percussion and auscultation will reveal that the margin of the opposite healthy lung is encroaching upon the sternum until the whole sternal region is thus occupied.

(*c*) The affected side becomes more markedly flattened, the area of cardiac pulsation more extended towards it, whilst the cardiac surface is being covered by the lung resonance advancing from the healthy side. The percussion note becomes duller and more wooden, and the hollow breath-sounds are less and less abundantly accompanied by râles. It is to be further noted, especially in left-sided cases, that the morbid pulmonary signs shift towards the upper axillary and infraspinous regions. In a word, the affected lung is shrinking towards its root.

(*d*) The range of temperature gradually contracts, and this restriction may often be observed, not only with regard to the high, but also the low register, which in the later hectic period of this disease is usually marked by subnormal points in alternation with pyrexial peaks (see Fig. 47). The other phenomena of hectic abate, the patient regains appetite and flesh, and in favourable cases the cough and expectoration

may cease, the cavities contract, and convalescence become fairly established.

It is to be noted that throughout the period of elimination and commencing cicatricial change, tubercle bacilli are to be found more or less abundantly in the sputum, which is highly purulent and nummulated. Nor can any safe inference be drawn, favourable or otherwise, in prognosis, from the abundance or paucity of the organisms, nor from their characters.

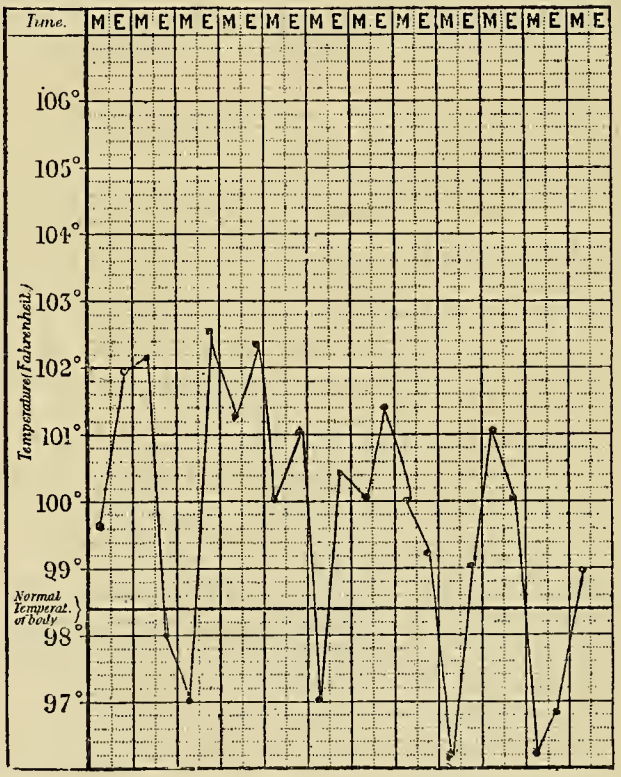

FIG. 47.—HECTIC TYPE OF TEMPERATURE ATTENDING THE RAPID SOFTENING AND ELIMINATION OF EXTENSIVE CASEOUS CONSOLIDATIONS. LUCY B.: LATE PERIOD OF ACUTE PHTHISIS.

In left-sided cases, when the lung has considerably contracted and all pulmonary signs have become quiet, the patient frequently complains of palpitation and occasional faintness, and ready disturbance of the heart's action. These symptoms are due to the heart being somewhat displaced, and to the fact that its anterior surface, unprotected by lung, is now in contact with the depressed parietes, while on the left it is further bounded by thickened lung instead of its normal elastic cushion of support.

Illustrative Cases—Case I.—As an example of the form of disease which we have been considering, we would refer to the interesting and instructive account of his own case published in 1901 by Dr. R. Mander Smyth.[2] In spite of the severity of the onset and the rapidity of its progress, the disease, under appropriate treatment, including a long stay at Nordrach, under the care of Dr. Walther, gradually became arrested. For twelve years Dr. Smyth was the head of a well-known sanatorium in England, and we are glad to add that, with one or two temporary set-backs, he has maintained his health—the lung disease, which had proceeded to excavation, remaining arrested—and is at the time of writing (January, 1920) wintering near Mentone. Latterly he has been troubled with gouty manifestations of a chronic type, affecting chiefly the left shoulder, and this complication may have exerted a not-unfavourable influence upon the tuberculous disease. As a personal experience of the variety of phthisis, which we are now discussing, described by a trained medical observer, the account will well repay perusal.

The notes of the two following cases illustrate further points in regard to this variety of pulmonary tuberculosis, and appear worth recording, though we regret that both terminated fatally after a period of improvement:

Case II.—T. W., aged thirty-six, Detective Sergeant in the City Police, was admitted into the Brompton Hospital under the care of Dr. Hartley on November 28, 1914.

He came of healthy stock, and there was no history of tuberculosis in his family. He had suffered from dyspepsia for some years, but had otherwise enjoyed good health. Since July, 1914, however, he had been feeling "run down," had latterly had a little cough, and in October on two occasions coughed up a small clot of blood. He continued at his work, but on November 9, when on duty in connection with the Lord Mayor's procession, he felt so unwell that he was obliged to leave his post and rest for awhile. Next day he felt chilled, but managed to continue his work until November 12, when he was admitted to the City Police Hospital, complaining of shivering and pains in his joints. He was found to have a temperature of 101°, and a few days later signs of consolidation were detected in the right upper lobe, and on November 18 tubercle bacilli were found in the sputum. On November 28 he was transferred to the Brompton Hospital. His early temperature record, commencing on the fourth day of his acute illness, was courteously supplied by the authorities of the City Police

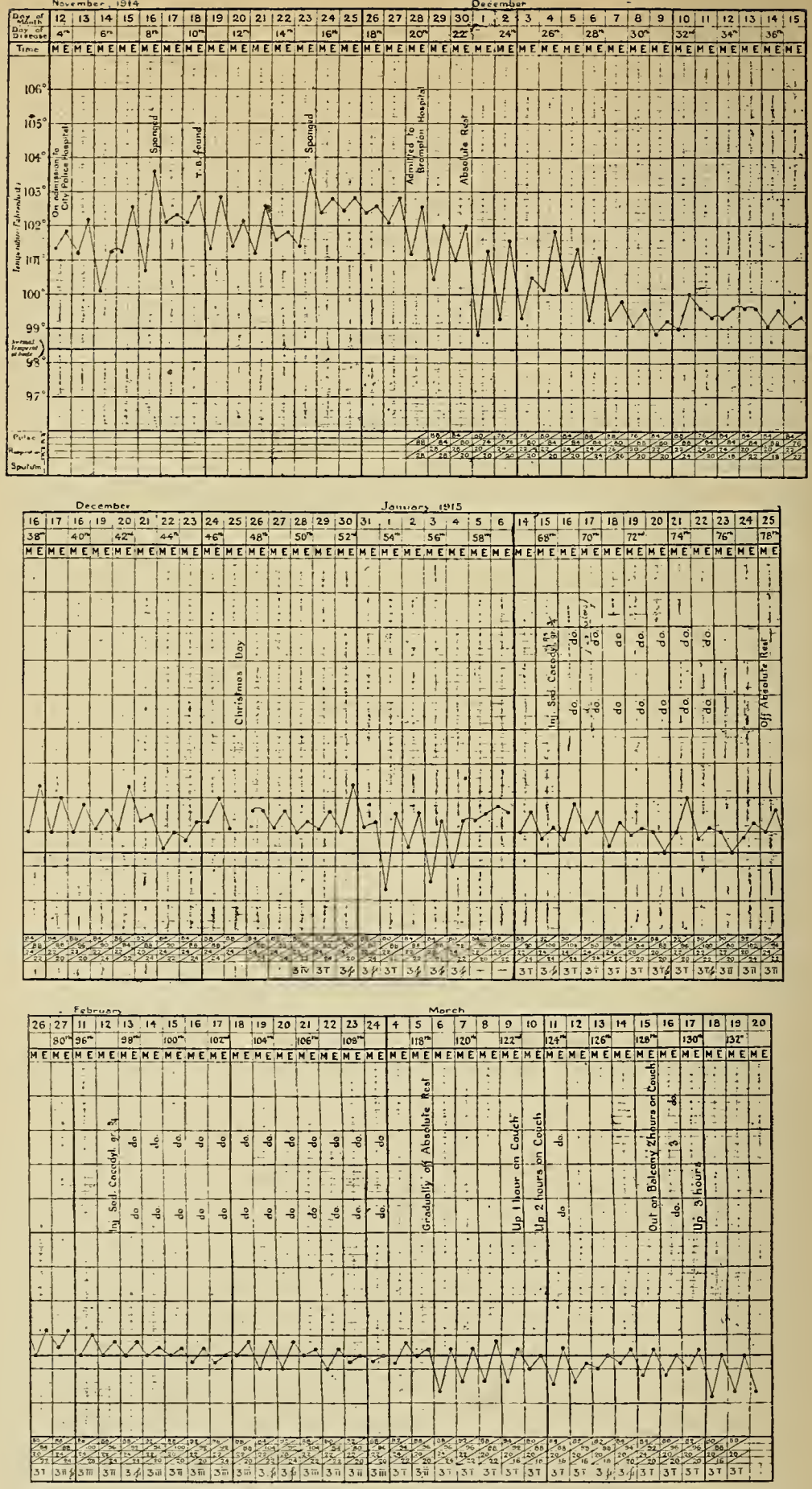

FIG. 48.—CHART SHOWING THE TEMPERATURE RECORD OF T. W., DETECTIVE SERGEANT, AGED THIRTY-SIX, WHO SUFFERED FROM ACUTE PNEUMONIC PHTHISIS.

Hospital, and is shown in the annexed chart (Fig. 48). It will be seen to be continuously raised, but of somewhat remittent type.

On admission to Brompton on the twentieth day of his acute illness his temperature was 102·6°, pulse 88, respiration 28. His height was 5 feet 10¾ inches, but owing to his condition he could not be weighed on admission. There was, however, no history of loss of weight. Some night-sweating was observed, and his sputum was muco-purulent in character and contained tubercle bacilli. In the right lung there were no physical signs of disease, but in the left lung an area of consolidation was present, as shown by impaired note, bronchial breath-sound, and bronchophony, extending from the third to the sixth rib in front. Over this area consonating râles were audible. Posteriorly the note was impaired from the apex of the left lung to the angle of the scapula, and moist sounds were heard over this region.

The patient was placed upon absolute rest, but the temperature remained irregularly raised for the next week, and then became lower, but for many weeks it oscillated between 99° and 100°, and it was not until the middle of February, 1915, that it became normal (see Fig. 48). The physical signs did not alter materially for a considerable time. On December 16 friction was audible in the left axilla, but on December 30 it had disappeared. By the middle of February the signs of consolidation had diminished, the note over the left front was less impaired, and bronchial breath-sound was only audible at times. There were still, however, moist sounds over the front of the left lung, extending to above the clavicle. Posteriorly the signs were much as on admission, impairment of note with moist sounds being present over the upper half of the left lung. The cough was now slight and the sputum scanty, but tubercle bacilli were still present.

During January and February he was treated with increasing doses of creosote up to a maximum of 25 minims twice a day in two drachms of cod-liver oil. From January 15 to January 22 and from February 12 to 24 he received also a daily subcutaneous injection of sodium cacodylate gr. ¾.

The case had now taken a more favourable turn, and hopes of recovery were entertained. On March 22, however, he had a serious hæmoptysis, bringing up a pint of blood, followed by a rlse of temperature to 104°. The pyrexia lasted for a fortnight, and the temperature then remained irregularly raised for some weeks, and it was not until the early part of May that it again became normal. The signs in the lungs had now increased, moist sounds being audible at the left base and also over the right middle lobe and the adjacent part of the upper lobe. The note at the right apex was also now impaired. His weight, which before the hæmoptysis had risen to 11 stone 7 pounds, had now fallen to 10 stone 7 pounds.

In this condition he remained till June 30, when he was transferred to the Brompton Hospital at Frimley in the hope of further improvement. His temperature, however, soon rose again. He was confined to bed, suffered a good deal from dyspepsia, lost weight, and left for

his home near Shrewsbury on September 30, where he died a few weeks later in November, 1915, just a year after the commencement of his acute illness. It is possible that but for the hæmoptysis the improvement previously noted might have been maintained.

Case III.—Mrs. X., aged twenty-nine, married, but without children, was first seen by Dr. Hartley in October, 1911, in consultation with Dr. Rose of New Barnet.

There had been no history of tuberculous trouble in her family, but she herself had never been robust, and six years previously her left kidney had been removed for tuberculous disease, with secondary involvement of the bladder. The operation was successful, and she lost her symptoms and regained her health, remaining well until June, 1911, when she began to suffer from cough, with some muco-purulent expectoration. She did not think she was losing weight. Her height was 5 feet 3 inches; weight 8 stone 12½ pounds. There were no sweats or hæmoptysis. On physical examination the percussion note at both apices was found to be impaired: some rhonchi were audible over the lungs, but no added sounds were heard at the apices. There appeared to be no fever, and the sputum on two examinations did not reveal tubercle bacilli. A diagnosis of bronchial catarrh with arrested tuberculous lesions at the apices was made, and under treatment by expectorant mixtures and cod-liver oil she soon recovered her health, losing cough and phlegm and gaining in weight.

She remained well until March 2, 1915, when she contracted influenza complicated by bronchitis. The sputum was scanty and purulent, and contained mostly pneumococci. Tubercle bacilli, though searched for on two occasions, were not found. On March 14, 1915, she was seen at Hadley Wood by Dr. Hartley, in consultation with Dr. Evill, who was attending her in the absence of Dr. Rose on war service. Her temperature was still raised and of intermittent type, varying from 98° in the morning to 101° at night. The tongue was furred, there was some sweating, and the impairment of note observed in 1911 was still manifest at the apex of the right lung; the note at the left apex was now fairly natural. Signs of general bronchitis were audible over the lungs, and at the left base there was obvious impairment of note, with bronchial breath-sound and sticky râles. A diagnosis of influenzal pneumonia was made, and as Mrs. X. was holding her own, a hopeful prognosis was given. The temperature, however, as may be seen from the "average chart" (Fig. 49), which dates from the third day of her illness, did not fall, and was of a somewhat hectic type, and bronchial breath-sounds still remained audible below the angle of the left scapula. The question of a vaccine was considered, and for this purpose the sputum was examined by the late Dr. Stansfeld, who found no tubercle bacilli or influenza bacilli, but a bacterial flora among which streptococci now largely predominated. A streptococcal vaccine was prepared and three doses given without effect.

On April 17 the patient was again seen in consultation with Sir Thomas Horder and Dr. Evill. Her temperature still remained raised to over 101° at night, and her cough was irritable. The tongue, however, was clean and the patient looked fairly well. There were still a few signs of general bronchial catarrh, but the note at the right base showed now only slight impairment. At this base, however, many crepitations were audible after coughing. The patient's appearance, general condition, and the continuing pyrexia strongly suggested tubercle, and it was decided that a further examination of the sputum should be made, and on this occasion tubercle bacilli were discovered by Dr. Stansfeld in considerable numbers. The case, therefore, was now proved to be one of arrested tuberculosis of the apices, with a

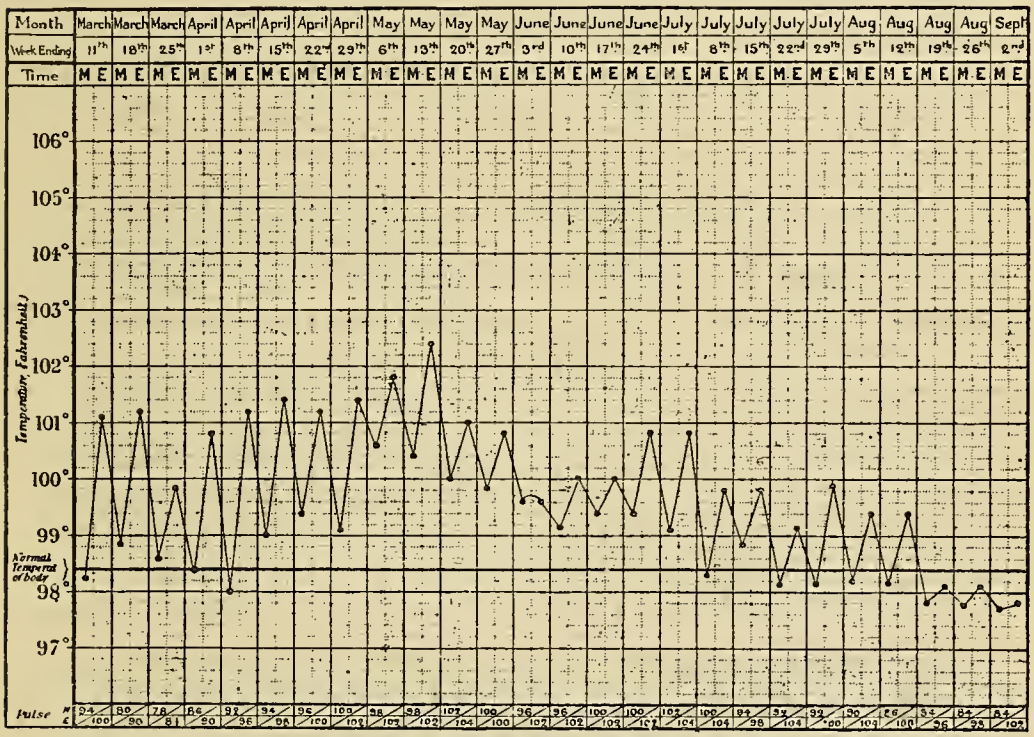

FIG. 49.—"AVERAGE CHART" SHOWING THE WEEKLY TEMPERATURE RECORD OF MRS. X, AGED TWENTY-NINE, WHO SUFFERED FROM ACUTE PNEUMONIC PHTHISIS.

(The record is obtained by taking the average of the morning and also of the evening temperatures during each week.)

recent acute invasion of the base of the left lung under the influence of an influenzal attack.

The patient was placed on absolute rest and treated on full open-air lines, and on April 21 was moved to a shelter built for her in the garden. From May 4 to May 20, June 3 to 12, and from June 23 to 29, she was given a daily subcutaneous injection of sodium cacodylate, gr. ¾, but the pyrexia still continued, though on a somewhat lower level.

On June 29 Mrs. X. was seen again in consultation with Dr. Evill.

By now the disease in the left lung had spread, and it was clear that the whole lung was involved, impairment of note and scattered crepitations being audible all over. The right lung was free from disease, except for the apical impairment of old standing. The cough was troublesome, the sputum purulent, and the fingers showed slight clubbing. The patient was placed on intensive iodine treatment, being given 20 grs. of iodide of potassium at 8 a.m., and an ounce of chlorine water in freshly prepared lemonade at 12, 2 and 4 p.m. Under this treatment the fever gradually abated and the temperature reached normal on August 17, having been raised for four and a half months.

On October 23, the temperature still remaining normal, the patient was moved to the Linford Sanatorium in the New Forest, and placed under the care of Dr. Felkin. Here, after a short stay, the temperature again began to rise and the cough and sputum increased. The case still appeared one-sided, and it was decided to attempt an artificial pneumothorax; but though six punctures were made, the attempt failed owing to the presence of widespread pleural adhesions.

On January 25, 1916, Mrs. X. was seen in consultation with Dr. Felkin. She was evidently much weaker, with hectic flush on the cheeks and a temperature of 101°. The pulse was rapid and soft, the sputum considerable in amount and containing tubercle bacilli in large numbers. A cavity, extending from the left clavicle to the fifth rib, was present in the upper lobe, and there was a second excavation at the apex of the lower lobe behind. The upper half of the right lung was also now invaded by active tuberculous disease. Under the circumstances it was obvious that no operative procedure could be considered, and that the patient's condition was one of great gravity. Garlic was prescribed without avail, and the patient died a few weeks later, just a year from the commencement of her illness.

This case illustrates the temporary improvement which may be observed in cases of this kind, and then the active extension of the disease in the other lung, which sometimes takes place and carries off the patient, even though the surroundings are all that can be desired. The failure to effect an artificial pneumothorax, owing to the presence of extensive pleural adhesions, will also be noticed.

The supervention of caseo-pneumonic tuberculosis, running a rapid course, was formerly not infrequent in *diabetes.* The statistics brought forward in 1883 at a discussion on diabetes at the Pathological Society[3] showed that about 35 per cent. of the cases terminated in pulmonary tuberculosis, most commonly in the second to the fourth years of the disease, and that in a still larger proportion—nearly one-half—pulmonary lesions were present. In the present day, when the death-rate from phthisis is about half what it was in 1883, such

figures are too high, nor does the complication, in our experience, now run as a rule such a rapid course in diabetic patients. Nevertheless, the possibility of the development of pulmonary tuberculosis must always be borne in mind when treating a case of diabetes, since in this disease the vitality of the tissues is lowered, and the patient becomes more prone to invasion by the tubercle bacillus. The lesions are more apt also to occur in situations other than apical, the base of the lung being not uncommonly attacked.

All pulmonary lesions in diabetes are not, however, tuberculous. In some having clinical features suggestive of phthisis, tubercle bacilli are not found, and the destructive lesion is a species of sloughing pneumonia.

2. Disseminated Form.

(1) **Florid Phthisis.** *Synonyms:* **Phthisie Galopante. Broncho-Pneumonic variety of Caseous Tuberculosis.**

In other cases, of happily rare occurrence, acute tuberculosis attacks both lungs, commencing simultaneously in many centres. This form of the disease, which is most common in young adults, and perhaps more frequent in women than in men, bears the same relationship to the preceding variety that disseminated bears to confluent broncho-pneumonia, and is characterised by the rapid softening of the caseous foci and by marked destruction of lung texture.

The symptoms are not essentially different from those of the more ordinary confluent form of acute phthisis which we have just described, but the dyspnœa is more urgent, and the progress to a fatal issue rapid, and as a rule unbroken. The flushed face, bright eyes, and alert mind contrast with the apathy, pallor, and prostration of acute miliary tuberculosis, and the physical signs at first of acute bronchial catarrh with the rapid development of numerous centres of consolidation, softening, and excavation, are equally characteristic. The rapid breaking down of the broncho-pneumonic centres, occurring first at the apices, is a striking feature, distinguishing this from more simple forms of broncho-pneumonia.

After death the lungs are found to present numerous areas of greyish-pink granular consolidation, with yellow caseous centres broken down into small cavities communicating widely with enlarged, more or less eroded, and acutely in-

flamed bronchial tubes. No miliary granulations of tubercle are to be seen, although the smaller yellow centres may at first sight resemble them.

Except on grounds of clinical accuracy, the distinction between acute disseminated phthisis and acute miliary tuberculosis is of little importance, for the prognosis in both is about equally fatal within a short period of from four weeks to two or three months.

The high and fluctuating temperature, hectic sweatings, purulent, sometimes blood-stained, and soon nummulated sputa containing elastic tissue and bacilli, in association with the physical signs, will render it impossible, except in the earliest stage, to confound this disease with acute bronchitis. In some cases, as in other varieties of rapidly progressing pulmonary tubercle, the temperature assumes the *inverse type* with evening instead of morning remissions.

An inquiry into the family history of a case of acute disseminated phthisis will frequently elicit evidence of a decided phthisical taint.

The following sketches illustrate the phenomena characteristic of this form of the disease:

Case I.—A woman aged thirty-one had had "inflammation of the lungs" two years previously, but had suffered from more or less cough, with frothy expectoration, for three years. She knew of no family predisposition to phthisis. Four weeks before admission into hospital she expectorated a small quantity, two teaspoonfuls, of blood, and the sputa continued to be tinged for five days. She had since suffered from night-sweats, emaciation, cough, and pain in the side and between the shoulders, of which symptoms she complained on admission. The pulse was 112; the tongue furred; catamenia regular. The physical signs on admission were harshness at the right apex, with subcrepitant râles; at the left, jerking breath-sound with prolonged expiration. She decreased rapidly in weight, losing 3½ pounds between October 26 and November 14.

On November 8 the physical signs were noted as unchanged. On the 21st she was much worse, had a red, tremulous tongue, a rapid pulse, great breathlessness, and much heat of skin. She could with difficulty stand from the trembling of her limbs and weakness. Subcrepitant râles were found diffused throughout the right side behind, with some defect in resonance not amounting to dulness. The temperature taken night and morning from this date showed a maximum morning height of 103°, average 101·6°; maximum evening temperature 104°, average 102·3°; difference between the average morning and evening temperature 0·7°. On the frequent occasions

when observed in the middle of the day, the skin was uniformly hot, and the pulse very rapid, usually about 120. Meanwhile the pulmonary physical signs advanced, the crepitations became more abundant, and extended through both lungs. There were signs of breaking down at the right apex, though the presence of a cavity could not be ascertained with certainty. On December 19 there were present "diffused blowing respiration, with sonorous rhonchus and scattered crepitations, more abundant at the bases, with some dulness; high temperature, and much dyspnœa." On December 15 the patient began to be troubled with diarrhœa, which continued more or less to the last. The emaciation and loss of power rapidly increased, the smooth red tongue became white with aphthous patches, and she gradually sank, having never evinced, however, any delirium or other morbid brain symptoms.

Post-mortem, the lungs were found studded with racemose groups of tubercle undergoing caseation and softening, and surrounded by ill-defined areas of pneumonia; the right apex was breaking up into small cavities. There was no miliary tubercle on the pleural surfaces.

The *continued* fever, the great and early prostration, the presence of physical signs at the apex, and later of diffused crepitations over the lungs, without any defined dulness, rendered the diagnosis of pulmonary tuberculosis being the prevailing lesion a tolerably certain one; but the case was differentiated from one of acute miliary tuberculosis by the early presence of marked pulmonary signs and a less degree of general prostration than often accompanies the latter disease, which is also attended by muttering delirium. There was, however, but little satisfaction to be derived from this reflection, for the prognosis was, so far as present knowledge could decide, inevitably fatal.

Case II.—W. A., van-boy, aged sixteen, was admitted into the Brompton Hospital under the care of one of us on August 18, 1913, complaining of cough, expectoration, and night-sweating.

There was no history of tuberculosis in his family, and he stated that he himself had been quite well until three months previously, when he began to suffer from cough.

On admission he was found to be febrile, having a morning temperature of 100·8°, which rose in the evening to 102·2°. His cough was troublesome, especially at night, and a good deal of yellow phlegm was expectorated, in which tubercle bacilli were discovered. The bowels were regular. The physical signs in the chest indicated invasion of the whole of the right lung, as shown by diminished mobility and general impairment of note, with scattered crepitations. Signs of excavation were also present below the clavicle. On the left side the percussion note was impaired from the apex to the third rib in front

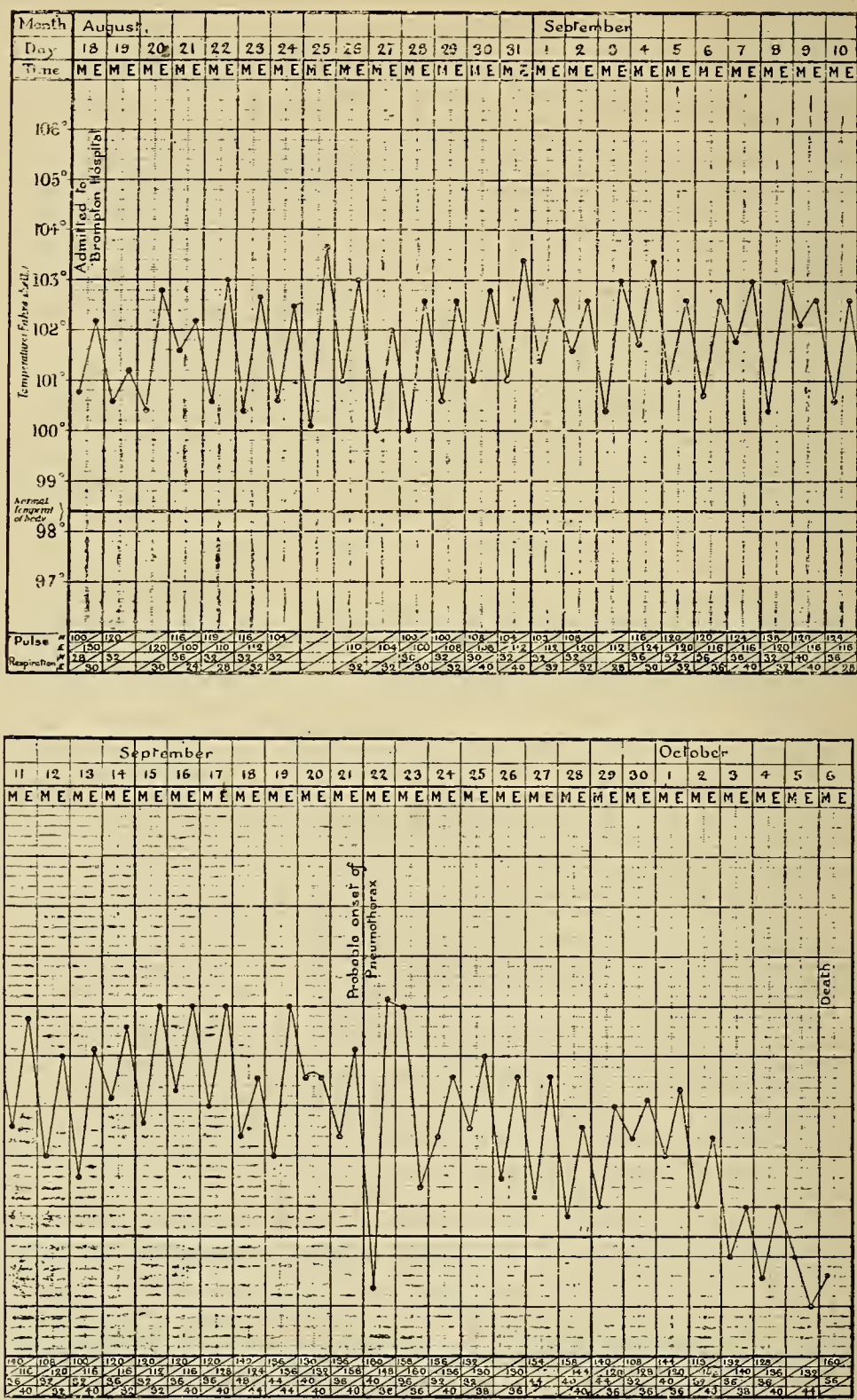

FIG. 50.—TEMPERATURE CHART OF W. A., VAN-BOY, AGED SIXTEEN, WHO SUFFERED FROM ACUTE CASEOUS TUBERCULOSIS OF THE BRONCHO-PNEUMONIC TYPE.

and over the upper third of the lung posteriorly, and over these areas sharp crepitations were audible.

After admission the pyrexia continued, the temperature, as shown in the annexed chart (Fig. 50), being of the high remittent type. The patient complained of some pain in the right side, but otherwise suffered little. His cough continued and he grew weaker. His pulse and respiration increased in rapidity, the former on September 19 being 136, the latter 44. Meanwhile the signs of excavation in the right lung were extending, and on September 19 a large thin-walled cavity, involving the whole of the apical portion of the lung and yielding a bell sound, was observed. A few days later he became cyanosed and dyspnœic, and some œdema of the feet appeared. The pyrexia continued.

On September 26 physical signs of pneumothorax of the right side were observed, its onset being probably indicated by a sudden fall in the temperature on September 22. On October 3 the pneumothorax had become practically complete, as indicated by a tympanitic note, bell sound and loud amphoric breathing heard over the right side of the chest. The loudness of the amphoric breath-sound suggested that the opening into the pleural cavity was large and patent, and such the autopsy proved to be the case. In addition to the air some fluid was also now present in the pleural cavity, as revealed by the succussion splash and movable dulness.

The temperature remained raised until October 1, when with increasing weakness it fell gradually to normal. The patient was now very dyspnœic and cyanotic at times, and died on October 6.

At the *autopsy* a pyopneumothorax was found occupying the right side of the chest, with the exception of its apical portion, where the pleuræ were adherent. The right upper lobe contained a large excavation, which communicated with the pleural cavity by a hole situated in the lower and anterior portion of the lobe, the size of a threepenny piece, and having thickened edges. Besides air the pleura contained 20 ounces of pus. In addition to the large cavity present in the right upper lobe, a second excavation of considerable size and containing much purulent material was found in the left upper lobe. The right middle and lower lobes were thickly studded with coalescing areas of caseous broncho-pneumonia, and in the left lower lobe similar, though less extensive, changes were observed. The larynx showed ulceration of the right vocal cord, and the trachea was extensively ulcerated. The bronchial glands were enlarged and caseous.

The case is an interesting one, as showing the rapid progress to a fatal termination which marks this variety of pulmonary tuberculosis—in this instance the duration being four and a half months. The ulceration of larynx and the occurrence of pneumothorax should also be noticed, since, as we have pointed out elsewhere,[4] these complications are rather

more common in the acute variety of the disease now under consideration than in ordinary chronic phthisis.

(2) Acute Miliary Tuberculosis.

The following case of miliary tuberculosis, the clinical features of which we will sketch, will serve to emphasise the fact that the tuberculous lesions of this variety of the disease run their course to a fatal issue with little or no softening, and also to illustrate the close clinical resemblance which may exist between some cases of acute miliary tuberculosis and typhoid fever:

Case I.—Elizabeth G——, aged thirty-four, a pale, sallow, grave-featured woman, with slight malar flush, was admitted into the Middlesex Hospital in March, 1885, complaining of cough and increasing weakness, with some pain of a pleuritic character in the left side, with which she had been troubled for three months. For a week she had been confined to bed with headache, cough, and some œdema of the feet.

On examination, only a few scattered bronchitic râles were discovered in the chest, the splenic dulness was increased, and the abdomen was observed to be full and tympanitic. The pulse was 100, small, feeble, regular; respirations 18; temperature 102·4°. The tongue was red and raw looking, and coated in the centre with a thin fur. No spots of a typhoid character were found on the abdomen, but numerous sudamina were observed scattered over the chest and abdomen.

The temperature ranged above 100°, daily reaching 102°, and frequently 103°, but was kept more or less modified by antipyrine, administered whenever it rose above 102°.

Up to April 20 no further chest signs were observed; there was then noted slight dulness and crackle after cough over the right subclavicular region, and some superficial crepitant râles at the angle of the right scapula. Diarrhœa was not present, but the abdomen continued prominent and tympanitic, and there was some tenderness on pressure, especially over the splenic region.

On April 25 the spleen was felt to extend below the costal cartilages. On May 7 there were signs of a little fluid in the peritoneum, and palpation of the abdomen gave a sense of soft resistance, as though from matting together of intestines by adhesions. Obscure crepitation was heard below the second cartilage on the right side, becoming more manifest and moister at the anterior base. On the left side some crepitations were heard in the mammary region. There were subcrepitant râles at both posterior bases.

The cough was troublesome, expectoration scanty and viscid. The sputum was carefully examined on several occasions without discover-

ing any tubercle bacilli. No diarrhœa was at any time present. The œdema of the legs increased, and the patient lapsed into a semi-conscious state, her temperature moderating somewhat, but keeping above the normal.

In this condition she lingered, until death ensued on June 30, three months after her admission.

The *post-mortem* examination revealed miliary tuberculosis of lungs, pleuræ, spleen, kidneys and peritoneum, with adhesive peritonitis. There was a small nodule of old disease at the apex of the right lung. None of the tubercles had softened. This patient's sister had ten years previously died of rapid phthisis.

Some of those who observed this case from time to time were doubtful as to whether it was one of enteric fever or tuberculosis. The continued fever, swollen abdomen, enlarged spleen and marked adynamia, were certainly suggestive of enteric fever, nor from experience could it be said that the absence of diarrhœa was sufficient to negative such a diagnosis. In both diseases, too, the leucocyte count in the blood is diminished. At the period at which this case was observed, the aid of Widal's test was not available. The temperature, however, although maintained at a high level, was of too fluctuating a type to correspond with the assumed period of typhoid. Moreover, the hectic flush is rarely, if ever, seen, and sweatings of sufficient severity to produce sudamina are, in our experience, equally uncommon in early typhoid. The pulmonary signs were at first quite compatible with either disease. As time went on it was apparent that an adhesive peritonitis with slight effusion was present, and the further development of chest signs rendered the diagnosis secure.

Cases are, on the other hand, sometimes met with in which the catarrhal pulmonary signs in the early weeks of enteric fever mislead to a diagnosis of acute tuberculosis, a mistake which, by preventing the strictness of dietary suitable for the former disease, may seriously hazard recovery. Enlargement of the spleen is an important sign in favour of enteric fever, since tuberculous disease of the spleen, although present in the case related above, is not usual. Later on in enteric fever the pulmonary signs fade, whilst the enteric phenomena become more marked.

It is to be observed that in the case which we have recorded, as in others of the same type, a careful examination of the expectoration for bacilli on several occasions gave a negative

result, a failure depending upon the absence of softening of the miliary tubercles in the lung. Occasionally, however, they may be found, as in an interesting case under the care of one of us, in which, though nineteen successive examinations of the sputum by Dr. Wethered proved negative, bacilli were yet found by him on the twentieth occasion, to disappear again on the twenty-first. On the death of the patient soon afterwards the lungs were found studded with miliary tubercles.

It is instructive to observe that the post-mortem examination of E. G. revealed some old-standing mischief at the apex of the right lung. Miliary tuberculosis is in truth a secondary disease, there having been most commonly in adult cases a previous abortive attack of phthisis, and when this is not so, some caseous remnant of a tuberculous lesion will be found elsewhere in the body. In children the primary focus is more often a caseous bronchial gland.

The following case is an example of that variety of the malady in which the pulmonary signs are more prominent:

Case II.—Mrs. L. D., housewife, aged twenty-eight, was admitted into the Brompton Hospital under the care of one of us on June 10, 1918, with the following history. She came of a healthy family, no members of which were known to have died of tuberculosis, and she had herself always been strong and well until the previous March, when she had a miscarriage. This was followed by abdominal pain in the right and left iliac regions, which did not, however, persist. In April she began to be troubled by cough, with some phlegm, and this was followed by nausea, failing appetite, night-sweats and wasting. Since May 13 she had attended the Out-Patient Department at the Brompton Hospital, and on May 21 tubercle bacilli were found in the sputum. For the twelve days preceding her admission to the hospital she had again complained of pain in the right iliac fossa, made worse by any movement.

On admission on June 10 she was found to be much emaciated. Height 5 feet 4 inches; weight 6 stone 13¾ pounds. She was flushed and breathless; temperature 101·6°; pulse 124; respiration 32. Her cough was very troublesome and persistent, and at times she suffered from distressing paroxysms of coughing, but sputum was absent or very scanty. Three days after admission a small quantity was obtained, and in it tubercle bacilli were again discovered.

The physical signs were interesting. The percussion note over the upper part of each lung, both anteriorly and posteriorly, was impaired, and moist sounds were audible over the upper and middle lobes anteriorly and down to the angle of the scapula behind. One or two râles were also heard over the base of the left lung. Pain and tender-

ness on palpation were present in the right iliac fossa. A vaginal examination showed nothing abnormal. In view of the physical signs, and also the finding on two occasions of tubercle bacilli in the scanty sputum, which, as we have seen, is rare in miliary tuberculosis, the diagnosis of acute caseous tuberculosis of the broncho-pneumonic type, rather than miliary tuberculosis, was made, a point, however, of academic rather than practical interest, in view of the grave nature of the prognosis in either case. The abdominal pain was thought to point to a possible appendicitis of tuberculous origin.

After admission the pyrexia continued, and, as shown in the chart (Fig. 51), was of the continuous type with some remissions. There

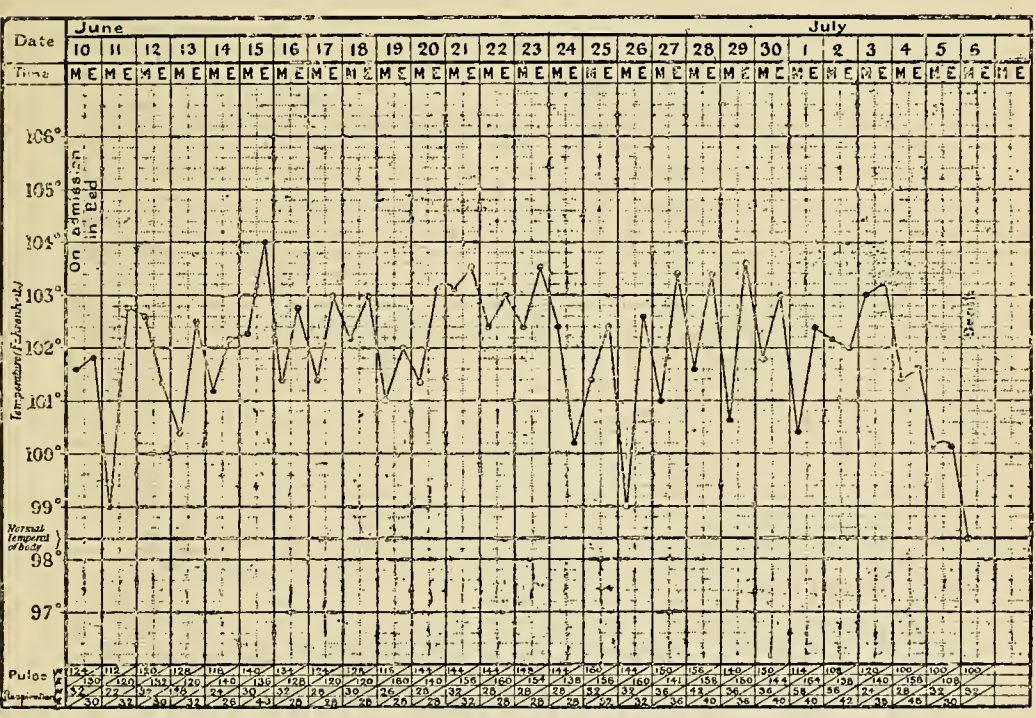

FIG. 51.—CHART SHOWING THE TEMPERATURE RECORD OF MRS. L. D., AGED TWENTY-EIGHT, WHO SUFFERED FROM ACUTE MILIARY TUBERCULOSIS AFFECTING THE LUNGS AND OTHER ORGANS.

was disinclination for food and some vomiting, but the abdominal pain gradually disappeared. On June 26 the patient, who was now very breathless and much weaker, complained of pain in the right side of the chest, and friction was detected over the lower and anterior part of the right lung. A few days later the heart showed signs of dilatation. On July 3 the friction had disappeared, but fine crepitations were now heard over the whole chest, both back and front, pointing to general involvement of the lungs. On July 6 the patient passed into a semi-conscious condition, the pulse became feeble, and she died the same day.

At the *autopsy* both lungs were found riddled throughout with miliary tubercles, yellowish in colour. Some recent pleurisy was visible

in the lower part of the right lung in front, accounting for the friction sounds heard during life. Miliary tubercles were present in the spleen, and in the peritoneum in the region of the cæcum, duodenum, and liver, with some peritonitis, thus explaining the abdominal symptoms which had been observed. There was no ulceration of the intestines or the appendix, and no focus of old disease could be discovered in the lungs. The bronchial and mediastinal glands were enlarged and caseous.

The duration of the disease in this case would appear to have been about three months. As no focus of old disease could be found in the lungs, it is probable that some of the larger caseating miliary tubercles softened and broke down, thus accounting for the presence of tubercle bacilli in the sputum during life. The blood-infection which caused the terminal miliary tuberculosis would appear to have originated in one of the caseating bronchial or mediastinal glands.

The *treatment* of acute tuberculosis of the lungs, as of the other varieties of phthisis, will be fully considered in later chapters, when the question of prophylaxis is also discussed.

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CHAPTER XXXIII

ON SUBACUTE TUBERCULOSIS OF THE LUNGS

In a certain not large proportion of cases of pulmonary tuberculosis the disease, instead of assuming the acute and rapid confluent or disseminated forms above described, follows a more insidious course, in which the destructive changes are effected by a drier and more gradual necrosis. In these cases great thickening of the alveoli, grey induration, in which some individual granules of tubercle may or may not be distinguishable by the unaided eye, is substituted for the more usual and rapidly caseating tuberculous process, although some points of caseation may here and there be observed. We have, in fact, a local pulmonary *tuberculisation* of slower and more insidiously destructive progress than caseous pneumonia, so far as the lung is concerned, but more obstinately and continuously invasive; more prone to be succeeded by early implication of the other lung, supposing both are not from the first affected; more quickly followed by disease in other organs, particularly the larynx and intestines; and, in short, though a subacute or chronic disease, yet one of more early average termination than the corresponding pneumonic form of phthisis. To this form of the disease we have in our former editions given the name of *pulmonary tuberculisation.* The condition is one intermediate between acute and chronic tuberculosis of the lung, and having some special features of its own.*

This form of tuberculosis spreads through the lung from apex to base, with a well-defined grey advancing margin, to which the highly vascular but crepitant lung tissue immediately beyond presents a striking contrast. On examining,

* A good illustration of this condition of lung will be found in Wilson Fox's *Atlas of the Pathological Anatomy of the Lungs,* Plate XVII., Fig. 1; but the figure is reversed, the disease being apical.

however, more minutely with a lens, the alveolar walls are found considerably thickened to some little distance (perhaps half an inch) beyond the defined margin, though the alveolar spaces are not occupied with catarrhal cells—at least, not uniformly so or to any material extent. A few outlying patches or nodules may often be observed, evidently of infective origin. The aspect and insidiously invasive character of the lesion most resembles lupus of the cutaneous surface; and since this comparison was first made in the 1878 edition of this work, lupus has been shown to be a truly tuberculous affection.

The following may be stated as the clinical features most distinctive of this form of phthisis, which is especially important from the difficulties attendant upon its early recognition, and on account of its relentless, although gradual, course.

The origin and progress of the disease are peculiarly insidious, with gradually increasing malaise and anæmia, nocturnal cough, and irregular fever. In many cases the temperature is persistently though slightly raised. In others it is at times elevated, during which periods there are fresh accessions of disease. The non-febrile intervals are of varying duration, the fever in this respect presenting no important difference from that which is observed in the more ordinary forms of phthisis, except that the completely apyrexial intervals are more rare and of shorter duration.

The physical signs are more characteristic. Instead of catarrhal signs proceeding to well-marked dulness and coarse crepitation or crackling, we find a continued weakness of respiratory murmur, with impaired expansion or actual flattening, while moist sounds may be altogether absent, or a few crackles may alone be elicited on cough. The percussion note becomes hardened, and we may be surprised by the appearance (having omitted to examine the patient for a week or two) of some feeble, blowing respiration, of hollow quality, still very dry, which increases in the same obscure way until an unmistakable cavity is present. This formation of a cavity by a process of dry necrosis is characteristic of pulmonary tuberculisation.

Huskiness of voice, or actual aphonia, is commonly one of the early symptoms in this variety of consumption. The huski-

ness may clear off, but the voice remains more or less permanently altered in quality. Too hasty a diagnosis must not, however, be made from this symptom, lest a grave prognosis be founded upon a simple laryngeal catarrh, although in many cases the larynx is actually involved. The digestive organs are as a rule affected early. The tongue may present a scanty white fur on a very red ground, with prominent red papillæ, an appearance which is significant of intestinal lesion, and still more so if the fur clears off in patches, leaving raw-looking glazed surfaces. Soon the symptoms characteristic of this lesion—alternating diarrhœa and constipation, with colicky pains, especially after food—make their appearance.

Patients who are the subject of this form of tuberculosis are usually of slender figures and good features, and among them are those more interesting examples of consumption or decline that novelists prefer to describe.* This variety is, however, amongst the more rare forms of phthisis.

The physical signs steadily, although, as already said, insidiously progress, and the average duration may be pretty safely reckoned as within two years of the first appearance of definite signs, although exceptional cases last longer, and it may be hoped that more may do so under modern methods of treatment. The intestinal or laryngeal complications cause great distress towards the last, and hasten the fatal termination.

The following case, which was seen by one of us in consultation at Harlesden, may be sketched as an example coming within the category of this variety of consumption:

Miss W——, aged nineteen, a typist, had been losing flesh for three or four months, with increasing weakness, which obliged her to relinquish business eight weeks ago. There was a history of an acute abdominal illness three years previously, with symptoms attributed to colitis, from which, however, she quite recovered. The only symptom complained of in her present illness, except the weakness and the loss of flesh, was relaxation of the bowels, which had been fairly constant, but kept in check by bismuth and occasional opiate medicines. The doctor stated that "the temperature had never been above the normal." The pulse had, however, always been notably quickened, 100 to 120. There had not been any cough beyond an occasional clearing of the throat in the morning, nor any expectoration. The catamenia had

* We may perhaps recall as examples Paul Dombey and "Smike" among Dickens's characters, and Marguerite Gautier of Dumas.

been absent for twelve months. The girl's aspect—her delicate features and complexion, with large lustrous eyes, long eyelashes, slight hectic flush, and notable emaciation—presented the features of what was formerly described as "decline" in medical literature and in fiction. There had never been any hæmoptysis. The stools had not been observed to contain mucus, nor to present any soapy, fatty, or other special appearances.

At the time of consultation—about 4 p.m. on December 16, 1910—the temperature taken for several minutes in the mouth was found to be 99·4°, and, on further questioning, the doctor who, owing to the patient's small means, had not had the advantage of a nurse to observe this symptom closely, admitted an occasional temperature of 99° or a little higher—a temperature often, but erroneously, regarded as "about normal." The pulse was quick and small; the abdomen was retracted, and over an area of some 4 square inches below the margin of the liver there was some tenderness on palpation, with slight resistance as compared with the left side, and impaired resonance on percussion. The margins of this resisting area were irregular and not sharply defined, and it was regarded as due to matted intestines and omental thickening. Neither liver nor spleen was enlarged. There was no swelling over the pancreas, no thickening or tenderness over the cæcum.

The thorax was small and the muscles wasted, and over an area in the left anterior axillary region, commencing at the outer third of the second rib in front, and extending downwards outside the nipple line to the level of the fourth rib, and backward to the mid-scapular line, the percussion note was dull. Over this region the respiratory murmur was weak and of a muffled bronchial quality, which in the mid-area became markedly cavernous, although still feeble; the voice-sounds and whisper here were pectoriloquous. But few râles were heard, only two or three coarse crackles. Elsewhere over the lungs on both sides the note was resonant and the breath-sounds weak but vesicular.

With great difficulty a small sample of sputum was obtained, watery in character, with a few opaque shreds. This was carefully examined before and after treatment with antiformin, but no tubercle bacilli were discovered.

In its peculiar insidiousness, in the obscurity of development of physical signs, and their notable dryness even until so near the end, and in the absence of associated catarrhal sounds and symptoms, this case is fairly typical of the form of tuberculosis now under consideration. So obscure were the signs and symptoms that, whilst one physician had a short time previously expressed the view that the lung was involved, two others had regarded the case as one of disease of the pancreas. There could be no doubt, however, from the present physical signs, as to the case being one of pulmonary

and abdominal tuberculosis, the lung being the seat of grey indurating tuberculisation, with dry necrosis, resulting in excavation at the centre, the site of the lesion being, however, somewhat unusual. The abdominal lesion presented the features of an intestinal matting, with tuberculous ulceration of the mucous membrane of the coils. It was obvious that a fatal termination might be expected within a few weeks.

At a second consultation, on December 31, the patient was notably weaker, the area of pulmonary consolidation had extended, and a few more liquid râles could be heard. She had had an acute attack of pain in the upper right abdominal region, and the tenderness here was more marked. The tongue was red, smooth, with some aphthous points. Some sputum was again obtained with difficulty; it was of mucoid, frothy character, and on examination tubercle bacilli in fair numbers were found irregularly distributed through it. The patient died on January 11, 1911.

There are two points to be noted further in this case. In all probability the temperature on more accurate observation would have been found to present a daily rise to 99° or 100°. No doubt, too, the abdominal complication, as usual, helped to obscure the chest signs; but involvement of other organs, whether of larynx or intestine, is a specially common feature in this variety of phthisis.

CHAPTER XXXIV

CHRONIC TUBERCULOSIS OF THE LUNGS

The subject of this form of tuberculosis has usually been depressed in health, through tardy convalescence from some other disease, bad living, mental anxiety, or overwork; he has had a persistent, though it may be a slight, cough for a longer or shorter time, and has been losing weight. Pyrexia is one of the earliest and most important signs that the pulmonary trouble is more than that of a mere catarrh, and hæmoptysis, however slight—even, it may be, only a mere streak in the morning sputum—is of serious significance. The pyrexia may not amount to more than a slight evening rise of temperature, but it is attended with malaise and increased cough at night.

At this stage the physical signs are but slight. The percussion note at one apex is slightly impaired, with perhaps some diminution in the width of the normal isthmus of resonance above the clavicle (Krönig's sign). The respiration is also weaker, and the inspiration wavy, or even jerking. There are usually a few rhonchi present, which, if limited to that apex, are very significant; and, in addition, there is heard at the extreme summit of the lung (supraclavicular or supraspinous region) a peculiar crumpling sound at the moment of cough, which differs both in time and degree from the crepitant sound audible at a somewhat later stage with the first inspiration following a cough.

These physical signs—and their early recognition is of much importance—are those of a broncho-alveolar catarrh limited to one apex. Such an apical catarrh is not necessarily tuberculous in nature, but should always be regarded with the gravest suspicion, and when taken in conjunction with symptoms of emaciation, quick pulse, and evening pyrexia, affords strong evidence of early tuberculous disease.

An immediate examination of the sputum for tubercle bacilli

is now imperative, and, if negative, should be repeated on at least two occasions, and with the help of concentration methods (p. 574). The importance of such an early and thorough examination of the sputum, and especially of that portion coughed up on first waking, in all cases in which there is the slightest suspicion of tubercle cannot be too strongly emphasised, for, as we shall see, our chance of arresting the disease varies to a great degree with the stage at which it is recognised, and proper treatment commenced. Too much stress also must not be laid upon a negative result, in the presence of the symptoms mentioned, unless the sputum has been examined on several occasions. In this connection we may refer again to the case of miliary tuberculosis of the lungs which we have already described, in which, with simple staining, tubercle bacilli were first found in the sputum on the *twentieth* examination. In children and others who swallow their expectoration, it is sometimes possible to discover the bacilli in the stools (p. 574).

In the early stages of phthisis a blood-examination shows, as a rule, some degree of anæmia, the red corpuscles being diminished and the colour index lowered, though not to the degree met with in chlorosis. Leucopenia is usually also present, the diminution in the number of leucocytes affecting chiefly the polymorphs, so that a relative increase of the lymphocytes occurs. As the disease progresses, and cavitation and hectic ensue, a leucocytosis is often observed, possibly reaching 20,000 or more, the polymorphs becoming increased. In the opinion of some observers a total increase of the lymphocytes is at this stage a hopeful element in prognosis.

Arneth has drawn attention to a change in the blood picture occurring in phthisis, as well as in other infectious diseases, and which is indicated by an increase in the blood in the number of neutrophile leucocytes containing one or two subdivisions of the nucleus, and a diminution of those with three or more. This change occurs early in phthisis, and tends, though with some curious exceptions, to become more marked as the disease advances and to lessen as the patient's condition improves. As Dr. H. A. Treadgold[1] shows in his interesting paper, in which he gives the results of his observations at the Brompton Hospital, further investigation is required before we can decide upon the value of

the Arneth count in the diagnosis and prognosis of pulmonary tuberculosis, but for the present we may accept that while the persistent presence of the Arneth blood-picture in marked degree would indicate a serious condition of the patient, its absence does not necessarily warrant a good prognosis.

In the acute stages of phthisis the systemic blood-pressure is commonly lowered,[2] ranging often between 90 and 100 mm. Hg. This is mainly attributable to depressed cardiac function from excessive absorption of tuberculous toxine, a similar lowering of blood-pressure having been noticed after a suitable injection of old tuberculin. When the disease becomes chronic and quiescent a recovery to normal systemic pressure may often be observed, and is of good augury in prognosis. We may add that a raised blood-pressure is no bar to infection, and we have known cases of arterio-sclerosis with high blood-pressure develope chronic pulmonary tuberculosis.

The disease tends insidiously to progress. The malaise, anæmia, nocturnal cough, and irregular fever increase, the physical signs, at first so obscure at one apex, gradually advance, and the other lung rarely escapes involvement. In a certain proportion of cases, probably larger than is generally supposed, the larynx becomes affected.

So far as the pyrexia is concerned, there is nothing characteristic to describe. During periods of activity the temperature is elevated, reaching, it may be, 100° or 101° at night, and falling to normal in the morning. Possibly also for a few days it may take on the *inverse type*, the morning record being higher than the evening; but, as we have already pointed out (p. 488), this as a rule occurs only in the rapidly progressing forms of the disease. The non-febrile intervals are of varying and sometimes of long duration.

The main features of an average case of this form of consumption, its physical signs, and some of its ætiological and other factors that have a bearing upon prognosis, are illustrated in the following sketch. It will be observed that the patient comes under observation, not at the commencement, but, as such cases so often do in practice, at a fairly pronounced although still comparatively early stage of the disease.

A tall, thin, worn, anæmic woman, aged twenty-nine, came to the hospital for advice on account of lung symptoms. She was suckling a child seven weeks old, with which she had become pregnant eleven months after the birth of twins, and whilst still suckling the survivor of them. Since her last confinement she had been suffering from increasing debility, shortness of breath, loss of flesh and cough, with yellow expectoration, recently tinged with blood. There were also occasional night-sweatings. Her chest was narrow and somewhat flattened, with deficient general mobility, but without any local depression or restraint of movement. The resonance at the left apex was impaired, the respiratory sounds there being harsh and wanting in vesicularity, and attended with some moist crepitations down to the second rib. Elsewhere the breath-sounds, though wanting in power, were of vesicular quality. The pulse was quick and weak, appetite indifferent, but digestion fairly good.

The patient inherited a disposition to consumption, having lost her mother from it when she was two years old; her sister had also suffered from an early stage of the disease.

She was unable to leave her home, and could only be induced to partially wean the child. Under tonic treatment, however, with cod-liver oil and a few lozenges to relieve the cough at night, she decidedly improved, gaining in health and strength, and putting on flesh rapidly. A month later the moist râles were no longer audible, except after cough, which elicited a few crackles. There now appeared a slight degree of flattening below the left clavicle, more distinct on deep inspiration. The respiratory sounds were feeble, while on the opposite side they were exaggerated, and careful percussion defined the margin of the healthy lung as extending slightly beyond the median line at the upper sternum. There was no evidence of a cavity at the left apex, nor of any extension of the area of disease. The pulse was quiet but weak, the appetite improved.

Such is a brief account of an average case, which suggests the following considerations:

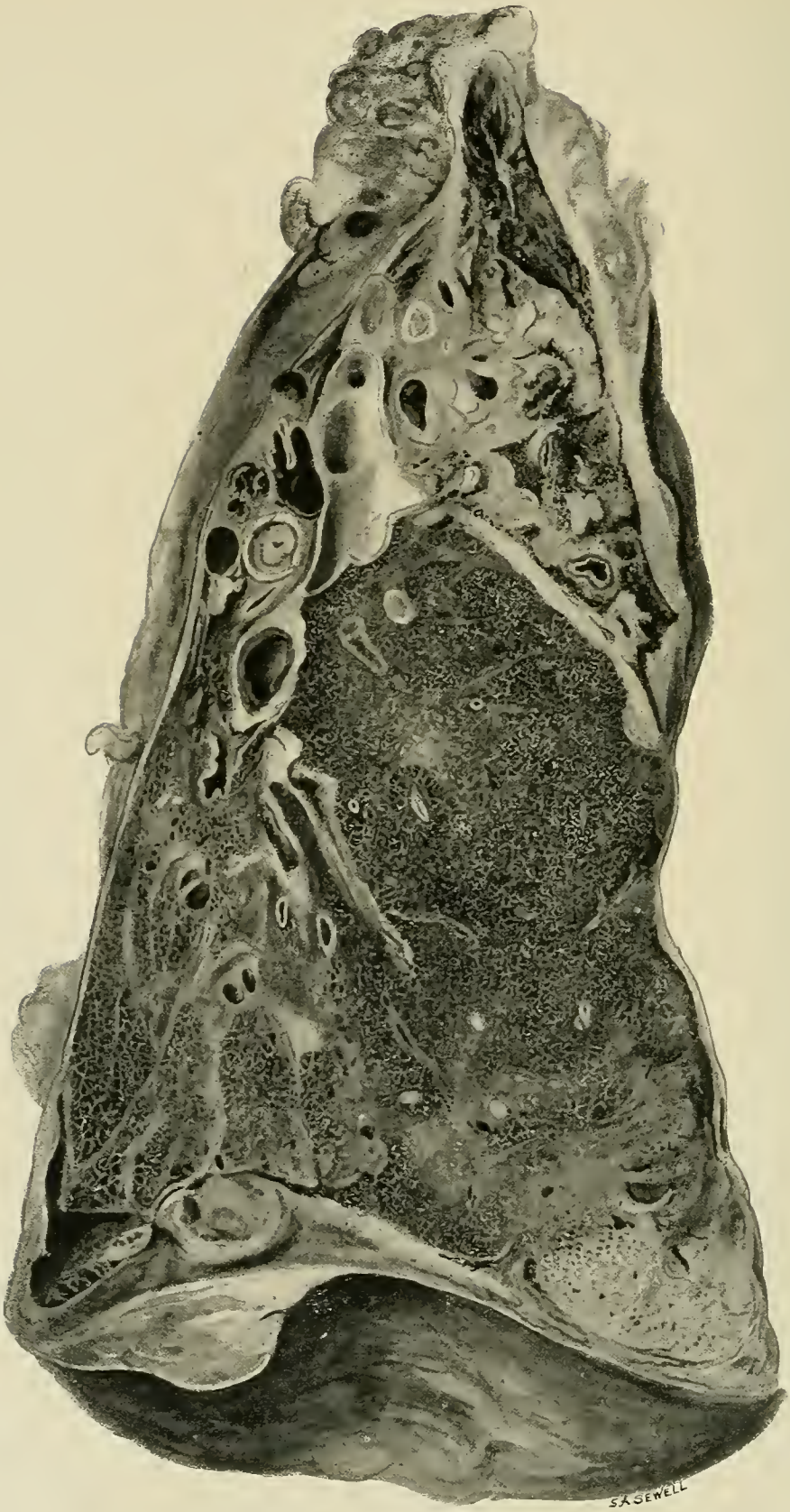
(1) The patient, with a phthisical family history, debilitated by adverse conditions—in this case rapid pregnancies and depressed circumstances of life—presented herself, suffering from a wasting illness of three months' duration, attended with definite chest symptoms. We can but rarely, at the moment of first seeing such a patient, know the range of temperature or whether there be tubercle bacilli in the sputum; but the history of night-sweatings and wasting would assure us of the one, and the character of the physical signs renders an examination for bacilli in such a case of less importance, for these signs reveal a definite lesion at the apex of the lung, which, under such circumstances, is almost always tuberculous.

(2) After a few weeks' treatment, even under the adverse circumstances of a case only treated as a hospital out-patient, a notable improvement was observed in the general condition of the patient, and two pulmonary signs presented themselves of clinical importance in the same direction. These were (*a*) the fact that, with diminished crepitations, showing a lessened activity of the disease, a commencing flattening and an impaired mobility of the chest over the part affected was observed; and (*b*) that, on careful percussion, to define the margin of the healthy (right) lung, it is found to extend towards the affected side up to and a little beyond the median line. These two signs—late flattening, coincident with lessened activity of disease (as contrasted with early flattening from sheer loss of lung substance), and compensatory expansion of the sound lung encroaching upon the region of the affected side—are of considerable value in favour of the hopeful prognosis of the case.

(3) The patient's history illustrates an important point in ætiology, the fact that a lowered resistance favours the attack, and reminds us of another important point in prognosis, namely, that the more unfavourable the circumstances which have led up to the illness, the better prospect is there of recovery if they can be removed or mitigated. "It is true that privation, excess, errors in habits of life, the sedentary occupations, the pernicious influence of certain trades, grief, anxiety, and the other wasters of vital powers, will not suffice to induce consumption in all, or even in the greater proportion of individuals; for these agents, so universally prevalent, are part of the daily lot or of the daily errors of many more than fall victims to consumption. But it is also true that, if to any or all of these conditions that of inherited tendency to phthisis be superadded, very few indeed escape the disease."[3] This remark is well borne out by the above, amid numberless other cases which must be familiar to physicians, although we now know that the acquired or inherited impairment of resistance operates by increasing the receptivity to a definite infection which may have the average wide distribution to which all are exposed, or, again, which may be more concentrated by general insanitation or specifically infected surroundings.

Most cases of "cured" early-stage phthisis are of the kind

PLATE XXIX



ARRESTED TUBERCULOSIS.

To face p. 507.

ARRESTED TUBERCULOSIS

The drawing shows the posterior half of the left lung. The upper lobe is seen to be considerably shrunken and converted into a dense fibroid, partly pigmented mass, containing one or two very small cavities, in one of which a little calcareous matter was found. No recent tuberculous disease is seen. The right lung also showed arrested disease—in the upper lobe a contracted cavity the size of a filbert, surrounded by fibroid material; and in the lower lobe some scattered pigmented tubercles of old standing, together with a small calcareous patch at the apex.

From a man aged fifty-one, who died from acute bronchitis, complicated with aortic regurgitation and extensive atheroma of the aorta. The tuberculosis in the lungs had undergone complete arrest.

(From the Museum of the Brompton Hospital. $\frac{5}{6}$ natural size.)

PLATE XXIX

above related. The arrest of the disease may be of long or even permanent duration, but the lesions which remain in the lung, although quiescent, still contain entombed within them organisms capable of fresh germination should the conditions again prove favourable. Hence the previous health-history of the patient helps us much in the prognosis in each individual case. Those cases in which the pulmonary delicacy is distinctly inherited are the least hopeful; those, again, in which the attack has been most distinctly provoked by adverse conditions of a definite and remediable kind are the most favourable. Of course, in each case the extent of lung involved and the intensity of the disease, to be ascertained only by physical examination, must, as already pointed out, most materially enter into the question as to outlook.

The patient whose history we have been considering suffered a second attack after the lapse of some months, with extension of disease, involvement of the other lung, and ultimately a fatal termination. Such is the average tendency in cases of this type, a disposition to arrest and to recurrence. It must be here remarked, however, that with arrest of disease a certain degree of immunity is gradually established, and provided the resistance be not broken down by premature return to the vicissitudes of current life this immunity may result in complete ability to withstand the test of acute influenzal and other illness.

Transition from Ordinary Chronic to Fibroid Phthisis.—All cases of chronic pulmonary tuberculosis present some evidence of repair, some attempt at arrest of the disease by the formation of protecting zones of fibrous tissue (Plate XXIX.). In patients in whom the natural resistance is great, this fibrosis may become a marked feature, and in extreme cases may warrant the appellation "fibroid phthisis," a condition which we shall presently consider. It must be remembered, however, that such fibrosis constitutes but a very imperfect form of repair. It is, in fact, the replacement of a higher by a lower form of tissue, which may be regarded as cicatricial, and which often proceeds to bring about deformities and other morbid conditions of its own.

The following case exemplifies fairly well the transition stages between simple chronic pulmonary tuberculosis and

fibroid phthisis, a transition pathologically easy, and clinically often to be observed:

John B——, aged twenty-nine, a butcher's assistant, came under the notice of one of us as an out-patient at the Brompton Hospital. He was a broad-chested, powerfully-made man, of medium height and florid complexion. He had led a rough but sober life, having followed his present business, which included the slaughtering of animals, for some years in Australia, and had enjoyed excellent health until shortly before Christmas, when, after getting wet, he caught a severe cold, which was followed by a cough, which had since increased, uninfluenced by treatment. Up to and at the time of his attendance in the following March he was still continuing his employment, but he now did so with difficulty, complaining of his cough and of increasing weakness, with decided emaciation. His father had died of consumption, brought on subsequent to the patient's birth by intemperance; there was no other hereditary tendency to the disease.

His chest, as before said, was broad and well-formed, without flattening or obvious impairment of expansion. The heart's apex-beat was in the natural situation. At the left clavicular and subclavicular regions the percussion note was dull, the dulness extending to the fourth rib; posteriorly the resonance was defective at the left supraspinous fossa. Scattered over the dull regions there was coarse crepitation, mingled with a larger humid crackle. These moist sounds were abundant, and masked to a great extent the respiratory murmur, which was decidedly harsh, but not distinctly bronchial. Its vesicular quality became gradually restored as the stethoscope was passed downwards. At the posterior base there were some scattered sibilant râles. On the right side the percussion note was good, and the breath-sounds were natural.

The physical signs at the present stage showed consolidated lobules of blocked alveoli, which were softening with varying degrees of rapidity, the coarse crepitation answering to the redux crepitation of pneumonia, the larger click being due to more profound destruction of tissue (softening). On the occasion of his first visit the pulse was quick and the tongue red, and although there was no elevation of temperature at the moment, it is probable that it rose slightly towards evening.

The patient was treated with an alkaline mixture containing small doses of iodide of potassium, and with cod-liver oil. A few weeks later the expansion of the left side of the chest was found to be decidedly impaired, the dulness had increased in hardness, but not in extent, and was very marked, especially between the left margin of the sternum and the mid-clavicular line.

In the space marked out by these two vertical lines (left sternal and mid-clavicular) the respiration was extremely feeble, and not attended with râles; the heart's impulse was diffused upwards to the second interspace, though the apex was only half an inch higher than natural.

To the left, again, of the mid-clavicular line the respiration was still feeble, and the râles much diminished, the dulness being somewhat greater than before. At the apex posteriorly there was bronchial respiration with imperfect pectoriloquy; the bronchial râles at the base had cleared up. The resonance of the right lung extended to the left margin of the sternum.

The physical signs showed—

1. That the disease had not extended; on the contrary, the signs of bronchial irritation at the base had cleared up.

2. That a wasting of the parenchyma of the lung had taken place; a small cavity had formed at the apex, with collapse and agglutination of air-cells elsewhere, thus causing a considerable reduction in the bulk of the lung and retraction of its anterior margin *from the median line;* so that between the left sternal line and a line drawn from the point of junction of the inner and middle third of the clavicle to the apex of the heart there was probably at this date no lung at all.

3. That an encroachment of the enlarging right lung, a slight shifting of the heart to the left, and a flattening of the chest wall had ensued to make up for the lost space. The flattening was, however, as yet very slight, and not noticeable until the patient drew a breath. The man had powerful parietes, and in such cases the displacement of heart and encroachment of the opposite lung precede, often for a long time, any obvious flattening.

It was remarkable with what rapidity these changes were taking place, and there can be no doubt that, after considerable lung destruction, the connective tissue of the bronchial, perivascular, and pleural sheaths was undergoing rapid development, and that the case was, in fact, lapsing into an early stage of fibroid phthisis. That the disease was not yet arrested seemed probable from the patient still losing slightly in weight and becoming more anæmic; but it had clearly become limited.

During the next month (May), although taking an acid preparation of iron, with a little quinine, and the oil, he lost two pounds in weight. Notwithstanding this, he had improved generally; the cough and expectoration had diminished, and he felt stronger. On June 8 he was still better, and had regained one pound since the last report. He had very little cough; all moist sounds had disappeared except a slight pleuritic râle on cough at the outer side and a little above the left nipple. He steadily improved up to August, five months after his first attendance at the hospital, when he was lost sight of.

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CHAPTER XXXV.

CHRONIC TUBERCULOSIS OF THE LUNGS—*Continued*

Fibroid Phthisis.

The term "fibroid phthisis" has been productive of much discussion. It was originally introduced by the late Sir Andrew Clark[1] to "embrace all those cases, whether local or constitutional, which are anatomically characterised by the presence, in a contracted and indurated lung traversed by more or less dilated bronchi, of fibroid tissue and of a tough fibrogenous substance, together with cheesy deposits or consolidations, and usually small cavities, commonly found about the middle and lower parts of the affected organ." The term is such a neat, concise and clinically useful one, that it has been generally accepted, with some reservations as regards the strict pathology of the disease as originally enunciated by its author. Indeed, at the present day its use is restricted to cases of pulmonary tuberculosis of an extremely chronic type, in which the resistance of the patient is considerable, and the formation of much fibrous tissue, with its attendant consequences, has resulted.

The prominent symptoms and signs by which cases of fibroid phthisis are distinguished are: increasing contraction and immobility of the affected side; traction of organs to that side; deadened percussion note and weakened breath-sounds of more or less bronchial quality, at parts intensely bronchial or cavernous; breathlessness; dragging pains; paroxysmal cough; occasional hectic, but general absence of fever; very chronic progress; long-continued one-sidedness of the disease, and correspondingly slow failure of nutrition. Such symptoms and signs bring the cases within the definition of phthisis, but phthisis of a special type.

The conditions presented post-mortem are those of a contracted, toughened, indurated, and usually pigmented lung, surrounded by a greatly thickened adherent pleura containing one or more rigid, dense-walled cavities, dilated bronchi, and cheesy encapsuled nodules.

On minute examination this condition of lung is found to have been produced by a growth of two kinds pervading its texture: (1) a connective-tissue proliferation, resulting in the formation of bands and processes of *fibrous* tissue, derived from the sheaths of vessels and bronchi and the subpleural and interlobular tissue of the lung; (2) a more important nuclear growth originating in a fibroid transformation of the individual tubercles, and resulting in the formation of broad tracts of *fibroid* tissue, which thicken the walls of the alveoli, compress, and finally efface them, unless they have been previously stuffed with their own inflammatory products. (3) In some small foci the process proceeds to slow caseation and softening. In other cases, as in that described in the previous chapter, a considerable destruction of the lung may have preceded the fibrosis.

The products of these processes become intimately mingled, but it is the tuberculous element which gives to the disease its peculiar clinical features, and renders the name "fibroid phthisis" applicable to it.

Numerous examples may be found of this somewhat inclusive disease, ranging from the most typical cases to those which are almost indistinguishable from ordinary chronic phthisis. We have described in the preceding chapter a case of simple chronic pulmonary tuberculosis, in which the transition into one of early fibroid phthisis was traced. It would not be difficult, however, to find examples in which the reverse takes place, the clinical characters of fibroid phthisis being gradually changed by subsequent activity of the tuberculous process, and all the features of the special variety becoming merged in the diffuse pulmonary destruction. Thus in a boy, to whose case allusion was made in a former edition of this work, the disease began with tuberculous destruction of a certain portion of lung, upon arrest of which the marked phenomena of pulmonary fibrosis supervened, and finally, again, active destructive changes set in, quite obscuring the fibroid characters, and resulting in death from pneumothorax.

The following case is a fair example of the variety of tuberculosis now under consideration:

George P., a sawyer, aged forty-three when he first came under observation as an out-patient at the Brompton Hospital, was a thin man, with dark hair, having no hereditary predisposition to lung disease, except that his father had suffered from "asthma." He had had slight cough for years, and in the preceding winter had been laid up for six weeks with "inflammation of the right lung." Since that time the cough had been continuous, and there had once been slight hæmoptysis. The cough was now paroxysmal, causing retching and often rejection of food; expectoration difficult, abundant, and of a pink tinge. He had lately been losing weight. The digestive functions were fairly good; the pulse a little hurried; there was no fever.

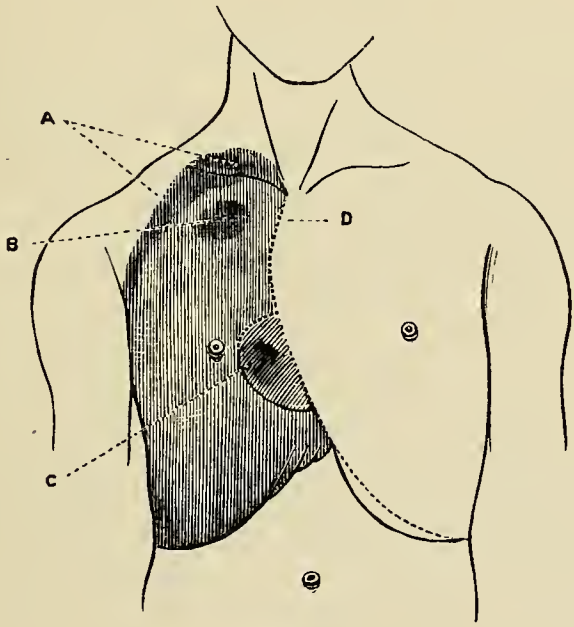


FIG. 52.

On inspecting the chest, cardiac pulsation was visible at the fourth right interspace (Fig. 52, C) to left of right nipple. This side was diminished in size and much restricted in movement, the intercostal spaces deepening with inspiration, while the left side expanded freely, with an uplifting movement of the shoulder. On careful examination, the apex of the heart was found a little to the left of the ensiform cartilage.

On percussion, the right side in the nipple-line anteriorly was dull to the second rib (A), comparatively resonant to the fourth, and below this point it was again toneless. The resonance of the left lung extended to the right of the sternum, as indicated in the diagram. The line of this resonance (D) sloped upwards to the episternal notch, being displaced in a downward direction by cardiac dulness at the fourth

cartilage. Hepatic dulness barely reached the costal margin. There was dulness throughout the axillary region and posteriorly from apex to mid-scapula, the note having a tubular quality in this latter region. Below the mid-scapula there was fair resonance to the ninth rib, though less and harder than on the opposite side; the lower two or three inches on the right side gave a flat note on percussion.

The percussion note over the whole left side, including the region of normal cardiac dulness and extending across the median line, as above indicated, was full and good in front and behind.

The auscultatory signs were in agreement with those of percussion. Above the clavicle on the right side the respiration was amphoric and dry; below the clavicle weak and bronchial to the base, with some rather large moist râles, friction and bronchophony. At one spot, corresponding with the second and third ribs in the nipple-line (B) the breath-sound was of tracheal quality, with scanty cavernous clicks and pectoriloquy. In the upper axillary region the respiration was amphoric, and the voice-sound pectoriloquous; in the supraspinous fossa and interscapular regions, cavernous-blowing with pectoriloquy. Blowing respiration extended to the angle of the scapula, where it became weaker and gradually annulled at the base. The vocal fremitus was generally increased on the right side. Respiration throughout the left lung was exaggerated and vesicular.

At the end of six months the patient had improved in flesh, appearance, and in strength, but complained greatly of cough, and expectorated much pink phlegm. Breath short on exertion; cough caused retching, but no rejection of food; appetite fair; digestion not very strong; bowels regular. Fingers observed to be clubbed.

The additional physical sign noted at this date was a distinct short systolic bruit at the point of maximum cardiac impulse, not appreciably increased by pressure nor confined to that spot, being also audible at the apex. Measurements of chest: from mid-sternum to nipple, right side, 4 inches; semi-circumference, $15\frac{1}{4}$ inches; expansion, $\frac{1}{4}$ inch. Left sterno-nipple measurement, $4\frac{1}{4}$ inches; semi-circumference, 16 inches; expansion, $\frac{1}{2}$ inch.

We may, by way of summary, aided by a glance at the figure (reduced as accurately as possible from a sketch taken at the time upon an outline diagram), interpret the above detailed physical signs as indicating at this period a general induration of the right lung, with much contraction, its anterior margin having receded considerably from the median line, exposing the pericardium, and having also shrunk away from the upper surface of the liver. Its upper lobe, and a portion of its lower, were extensively excavated, the cavities being old, tolerably dry, and shrunken with the general contraction of the lung. The pleura, judging from the hardness of percussion, feebleness of breath-sound, and fixity of chest

walls, was probably greatly thickened. The liver was drawn up within the costal margin, and the heart considerably displaced to the right, its axis being, however, but little altered.

His chest was repeatedly examined during the many subsequent months of his attendance at the hospital, but beyond some variation in the dryness of the sounds, there was no important change in the physical signs. The urine was more than once examined, but was always found free from albumin.

The left lung remained healthy, and though the patient continued thin and cachectic-looking, with a troublesome cough, he held his ground fairly well, and rather improved in general health. At times the expectoration would become very abundant, and occasionally of a pink colour, probably due to fresh irritation and slight sanguineous discharge from the walls of the old cavities. The most troublesome symptom throughout the case, and one which is common in greater or less degree to all those cases of tuberculosis in which indurated thick-walled cavities are present, was the paroxysmal cough terminating in vomiting, occurring especially after meals.

On ceasing attendance at the hospital, after a period of nine months, the patient, though not free from cough, continued for a time to improve, but he soon afterwards began again to emaciate, and the vomiting with cough returned. He again attended in January the following year for three months, and improved.

The above-related case represents very well the main features of fibroid disease of the lung. The indurative disease supervened presumably upon an acute tuberculous pneumonia affecting the upper part of the right lung, and manifested itself with tolerable rapidity, the characteristic symptoms and signs being fully developed within six months of the termination of the acute disease.

The question as to the rapidity with which this disease may advance is one of great interest, and requiring further observation. We cannot but think that, reasoning from the morbid appearances found in the post-mortem room, we are apt to regard such diseases as older than the clinical history will warrant us in believing; on the other hand, though it is probable that the fibroid induration of the lung may proceed with great rapidity to such a stage of shrinking as to produce marked clinical signs, its subsequent progress is very slow and difficult to measure, consisting mainly in the further hardening of an already indurated lung, the gradual widening of the bronchial tubes, and filling up of the loose œdematous areolar tissue between the separated pleural layers by dense fibrous

growth. We can readily perceive, therefore, how its earlier stages, which are attended with striking alterations in physical signs, may be passed through with comparative rapidity, while the later progress is necessarily slow and difficult to estimate.

No doubt the mechanical conditions of the cavities to which we have referred, and which render the removal of expectoration difficult, have much to do with the production of vomiting, and cause it to be a particularly common symptom in these cases; but the reception of food into the stomach has seemed to be in many cases so constantly followed by cough ending in vomiting as to render this mechanical explanation insufficient, and we have been led to attribute it to an undue reflex irritability of the pneumogastric nerve.

The following notes of a patient who had been under observation at the Brompton Hospital for more than forty years illustrate the great chronicity which this form of the disease may sometimes attain:

E. P. was admitted, at the age of seventeen, into the hospital, under the care of our colleague, the late Dr. Pollock, in November, 1877, for cough and weakness of seven years' duration, having also had slight hæmoptysis in 1874. On her mother's side there was a predisposition to phthisis, her maternal grandmother and uncle having both died of this complaint; and twelve years later, in 1889, her mother herself succumbed to it.

The physical signs on admission were, to quote Dr. Pollock's note, "on the right side dulness, flattening, and cavernous breathing, with gurgling and humid crepitations to the anterior base and in the axilla. In the supraspinous fossa some cavernous sounds were heard. The respiration was tubular in the interscapular region. Some crackles were heard at the posterior base. The left lung reached to the right border of the sternum; no abnormal sounds were heard over it." On January 17, 1878, the right lung was found to be dry, except for a few fine crackling sounds after cough in the infraclavicular region. The posterior base was clear.

In February, 1878, the patient left the hospital greatly improved, her weight now being 7 stone 3¼ pounds, a gain of 6¾ pounds since admission. After leaving, she remained fairly well for some years, though suffering on and off from cough.

In 1892 she again entered the hospital, under Dr. Mitchell Bruce, and again derived benefit.

On October 9, 1899, she came under Dr. Hartley's observation at the hospital, complaining that her cough had been worse lately and her breath shorter. Her general condition was fair, though her appetite was poor. Her height was about 5 feet 3 inches, her weight 6 stone

11 pounds. On examination, the *right* side was flattened with but very poor expansion, and yielded all over a markedly impaired note to percussion. The heart was drawn to the right side of the sternum, and pulsation extended as far as the right anterior axillary line (Fig. 53, A). Cavernous breath-sounds were heard over extended areas of the right lung, with scattered crepitations. The *left* lung seemed free from disease and to be hypertrophied, extending for about an inch to the right of the sternum (B). There was no clubbing of the fingers.

The diagrams (Figs. 53 and 54) show the condition of the chest, which was but little different from that observed by Dr. Bruce in 1892.

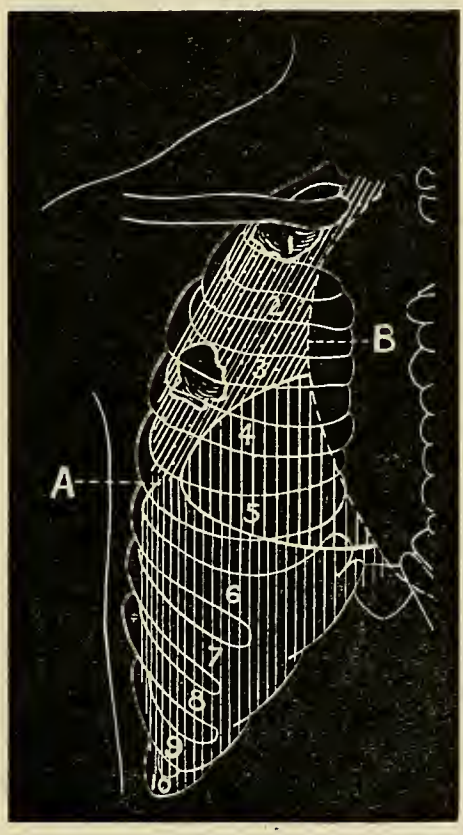


Fig. 53.

In June, 1900, she had improved in every way, although her cough continued. In January, 1901, her weight was still 6 stone 11 pounds, and her physical signs showed no change, except that now some crepitations were heard at the apex of the left lung posteriorly. In the expectoration, which was never very large in amount, a few tubercle bacilli were found.

From 1901 to 1906 the patient was under our observation from time to time, and, on the whole, somewhat improved. In May, 1903, her weight was 7 stone 6½ pounds, and in May, 1906, 7 stone 8 pounds. On the latter date crepitations were still audible at the left apex, but

showed no signs of extending. She was still able to carry on her occupation as a needlewoman.

Since 1906 she has lived in London, and has kept fairly well, though subject to bronchial attacks in the winter, when her cough and breathing become troublesome. Apart from these attacks she has had very little cough or phlegm. In 1910 she suffered from an abscess in the right breast, which her doctor regarded as tuberculous. It discharged for about twelve months and then healed.

On February 25, 1920, she came at our request to the hospital for examination. Her age was now fifty-nine. She looked well in herself, but was somewhat short of breath on exertion. Weight, 7 st. 2½ lbs.

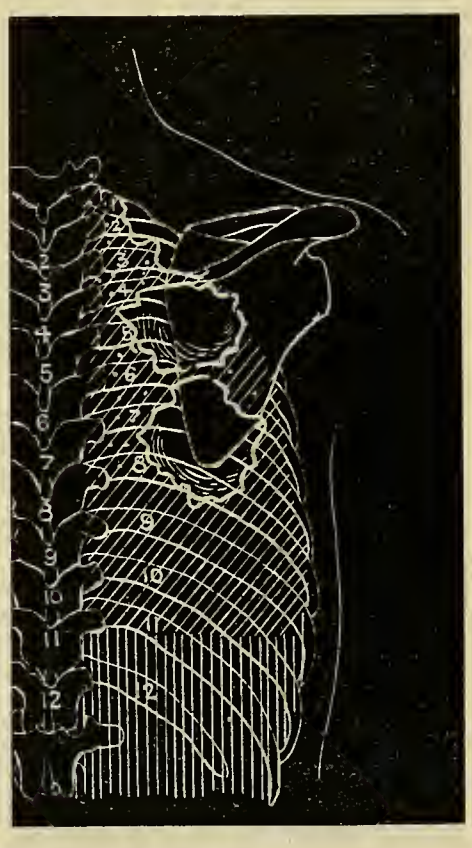


FIG. 54.

There was but little change in the pulmonary condition. The right lung was contracted and excavated, and the right side expanded but little on inspiration. The note at the left apex was still slightly impaired, and a few moist sounds were audible in this region, but there appeared to have been no extension of the tuberculous disease since her last appearance in 1906. The heart was drawn over to the right side as before, but the blood-pressure was now distinctly raised, measuring in the right brachial, in the sitting posture, 170 mm. Hg. The second aortic sound was rather forcible. The urine was of low specific gravity, and contained no albumin or sugar. The spleen and liver were not enlarged, and there was no

diarrhœa, or any evidence of lardaceous degeneration. Her sight was beginning to fail, and she was now unable to continue her needle-work.

This case is of great interest in that the signs have been traced, through a period of forty-three years, from an apical disease to one involving the whole lung and coming into the category of fibroid phthisis. In spite, also, of the long duration of the case, the affection of the opposite lung remains but slight. She appears indeed to have long attained arrest of the tuberculous disease, and for some thirty years to have enjoyed fair health, though possessing only one functionating lung.

Prognosis.—The outlook in fibroid phthisis varies with the somewhat diverse cases grouped together under this heading. The disease, however, may be said to be very chronic, and capable in a considerable number of cases of permanent arrest. There are, however, certain points to be taken into account in estimating the duration of a case. The cachexia may be marked, almost resembling that of some cases of cancer. The wall of a chronic cavity may be traversed by a large vessel, which, yielding on its unsupported side, forms an aneurism, and gives rise to recurrent or fatal hæmoptysis. Out of eight well-marked cases in which post-mortem examinations were made by one of us, hæmoptysis was the cause of death in two.[2]

The opposite lung in most cases becomes in course of time involved at the apex with secondary tubercle, but this is usually of the grey miliary type, and generally chronic or quiescent. This is exemplified by the case of E. P., above related, and also by that of a patient who was brought by one of us before the Clinical Society so far back as 1868. In association with chronic destructive lesion of the left lung, this patient had also distinct evidence of involvement of the apex of the opposite lung, and, in addition, a small quantity of albumin in the urine. He subsequently, however, led a rural life, and his health yearly improved. The right lung became greatly enlarged, the disease in the left remaining perfectly obsolescent, and the patient was in 1885 in good flesh and, to all appearance, in robust health.

In the later stages lardaceous changes in various organs, especially the spleen, liver, and kidneys, commonly supervene, and may lead to the death of the patient. Of the three most

characteristic cases in which we have made post-mortem examinations, in one there was extensive lardaceous degeneration of liver and spleen with granular kidneys; in another of the spleen only. Albumin in the urine, absent in the above-related case, is often the earliest clinical evidence of this change.

The condition of health and physique maintained by some patients is remarkably good. We have notes of a postman presenting exceedingly well-marked signs of this form of tuberculosis, who almost entirely lost his cough while attending the hospital, and was able to resume his duties, walking fourteen or fifteen miles a day; and we might mention some other patients capable of considerable physical exertion on level ground. Persons who for many years have had one lung dormant or "gone," as they usually describe it, are not uncommonly met with, and belong to this category. Mention has already been made of two such cases, and we can also recall, amongst others, that of a lady who for thirty years had her left lung similarly affected. This patient led a sheltered but useful life, and rarely suffered from pulmonary symptoms of any kind. Her chief complaint from time to time was of failure of heart's action, causing chilliness and a disposition to fainting, with occasional attacks of great cardiac oppression, which, but for the absence of any severe pain, would be described as angina. There was no evidence in this case of valvular disease or decided dilatation of heart, but the organ was uncovered by the retracted left lung, and the sounds were feeble. We have observed in many chronic left-sided cases of tuberculosis great functional disturbance, or rather irritability of heart, doubtless attributable to its being less supported and protected by lung than in health.

REFERENCES.

[1] "A Case of Fibroid Phthisis," by Andrew Clark, M.D., *Transactions of the Clinical Society of London,* 1868, vol. i., p. 188.

[2] For an account of these cases, see

(1) "Three Cases of Phthisis with Contracted Lung," by R. Douglas Powell, M.D. (Case I.), *Transactions of the Clinical Society of London,* 1869, vol. ii., p. 181.

(2) "Some Cases illustrating the Pathology of Fatal Hæmoptysis in Advanced Phthisis," by R. Douglas Powell, M.D. (Table, p. 58, Case I., F. W.), *Transactions of the Pathological Society of London,* 1871, vol. xxii., p. 41.

CHAPTER XXXVI

TUBERCULOUS EXCAVATION OF THE LUNG—THE CAVITY STAGE OF PHTHISIS

THE excavation stage of some cases of phthisis is so prolonged, and the symptoms are so decidedly grouped about the cavity, that at some schools it is the custom to name them cases of "cavitation." Whilst we do not regard such a term as admissible in any formal sense, however appropriate it may be as a colloquial expression in clinical teaching, there are yet many points in the diagnosis and treatment of different kinds or conditions of cavities which are worthy of consideration in a separate chapter.

We have seen that destruction of lung is the essential anatomical feature of pulmonary tuberculosis. In the most rapidly fatal cases the destruction takes place simultaneously at many centres, or involves such an extent of lung as to render hopeless any effort at repair or compensation. But in a large proportion of cases the disease affects principally one apex, the active symptoms attendant upon the pulmonary consolidation and softening after a time subside, the appetite returns, and the patient begins to regain strength and flesh. The cough still continues, however, and auscultation reveals the existence of a cavity at the apex concerned, the disease being now usually described as having advanced from the first (consolidation) through the second (softening) to the third (cavity) stage.

If these terms were strictly employed in a structural or anatomical sense as regards the lungs only, they would not be objectionable; but, in fact, they are too often extended in their application to the whole disease as it affects the patient, and therefore become fruitful of error and misunderstanding. These so-called stages of phthisis have reference merely to

the local effects of that disease, involving perhaps a fiftieth part or, it may be, a large portion of one or both lungs; they have no meaning as applied to the present or prospective duration of the disease. A man with a big cavity is frequently better off as regards life and health prospects than one with a "first stage" patch of disease no larger than the area of a shilling. A cavity once formed is so much lung gone, and it is for many reasons better that the irremediably diseased portion should be cleared out than that it should remain as a centre for fresh irritation, which may break down or infect the system at any time. Our anxiety as regards the immediate prognosis rests upon the condition of the outlying portions of the affected lung, and still more upon the degree of integrity of the opposite lung. Yet the student rarely looks beyond a cavity, upon the discovery of which he is apt to classify the case and to decree the fate of the patient. The physician, too, is often beset by anxious inquiries from relatives and friends as to the existence or non-existence of a cavity, upon which they base their hopes and fears, and upon his capacity to discover which his reputation is registered in their estimation. These terms, then, being inaccurate and misleading, should never be used in their general sense.

To resume, however, the special subject of the present chapter. Cavities may be considered under four heads: (1) the recent cavity; (2) the quiescent cavity; (3) the secreting cavity; (4) the active or ulcerous cavity.

1. **Recent Cavity.**—The recent cavity is the first result of the breaking down of caseous nodules in the lung. In cases of sufficient intensity these undergo softening and liquefaction in few or many centres. We do not, however, obtain any physical sign of the production of a cavity until communication is effected with a bronchus and some of the softened matter is expelled. From this moment we have cavities existing in the lungs and accessible to the air during respiration.

To yield the auscultation signs which are regarded as necessary for diagnosis, a cavity must have the dimensions of a walnut, or larger, and must communicate freely with a bronchus. But on comparing our clinical notes with post-mortem observations we shall find the former most commonly inadequate if we have awaited the presence of cavernous

breathing and pectoriloquy, and such-like orthodox signs, before admitting the existence of excavation.

The pulmonary consolidations break down into cavities in one of two ways, which are not, however, essentially different. In the first case, many minute lobular centres of softening are found which yield to auscultation moist crackling or humid clicking sounds; these increase in size and abundance as the softening centres extend and coalesce into larger cavities, until finally we have cavernous râles. The respiratory murmur which—bronchial with the first consolidation—had become weakened and more or less masked by the moist sounds, becomes again audible, but much altered in quality, assuming the more or less distinctly cavernous character. Whispering pectoriloquy now becomes marked, and in some cases a characteristic succussion of air may be heard on coughing, the so-called "post-tussive suction." On percussion, the note over a cavity is usually impaired owing to consolidation of the surrounding lung, but over extensive excavation with thin walls, tubular or amphoric resonance may be obtained (p. 39), with possibly the *bruit-de-pot fêlé*, if the cavity be near the surface, and if it communicates freely with a bronchus.

Perhaps the large majority of phthisical cavities form and increase in the way thus briefly sketched, but in some cases they are produced in a slightly different manner. It may happen that we fail to obtain distinct evidence of pulmonary softening for a time, when troublesome dry cough and hectic symptoms point strongly to its presence. There may be dulness, harsh breathing, and some fine spongy crepitation, increased after cough, but none of those distinct clicks characteristic of pulmonary softening. Then the patient will suddenly, in the course of the night, perhaps, expectorate a considerable quantity of purulent matter, and we find evidence —cavernous râles, etc.—of the existence of a cavity. The explanation of these phenomena is obvious enough; a nodule of consolidation of appreciable dimensions, but rarely exceeding a walnut in size, becomes uniformly caseous, and then softens in its centre and gradually liquefies throughout before communicating with a bronchus, when its fluid constituents are at once expelled, and auscultatory evidence of the existence of a cavity becomes abruptly developed. In the post-mortem room we may often cut through such softening

nodules in all stages of ripeness for exit; they sometimes undermine and rupture through the pleura, and may well be designated *caseous abscesses.*

The following case, alluded to by one of us in a clinical lecture delivered at the Brompton Hospital,[1] illustrates the rapid formation of a cavity at the right apex, and the cicatrisation and clearing up of signs and symptoms which may sometimes follow:

A. W., a pale, scrofulous-looking girl, who was in attendance at the time of the lecture, had been under observation at the hospital since March, 1872, when, at the age of fifteen, she first came as an out-patient. She had then a somewhat loud cough, which had troubled her all the winter, but there were no other definite symptoms and no discoverable pulmonary signs. Her family history was good, but she had been delicate since an attack of measles in childhood. She recovered on ordinary treatment, but returned in October, 1874, and again improved, but was ailing during the winter. In the spring of 1875 her principal complaint was of a painful affection of the left breast; she still had no definite pulmonary signs or symptoms beyond general delicacy and slight cough.

In June she had whooping-cough rather severely; and six weeks after she still whooped with the cough, which was attended with thick, difficult expectoration. On again examining her chest, one was somewhat surprised to find dulness, with marked cavernous breathing and some gurgling below the right clavicle; moist crepitant râles were also rather sparsely scattered over the posterior base. Fever and hectic symptoms were now marked. The prognosis appeared grave, and she was recommended to obtain an in-patient's letter. She did not come into the hospital, however, until January, 1876, and meanwhile improved as an out-patient. In the preceding October no moist sounds were audible, and on admission into the ward in January her chest was examined with great care, and on several subsequent occasions, but nothing beyond some harshness and feebleness of breathing at the right apex could be discovered.

After leaving the hospital in 1876 she remained well for a considerable time, and early in 1883 was married. In the course of that year she had a miscarriage, and afterwards suffered much from menorrhagia. In May, 1885, she again came under observation, with cough, especially during the night and in the morning, and some loss of flesh. She was nursing entirely a child eight months old. At the right apex, the seat of former mischief, there was slight dulness and some deep crackling and catarrhal râles, but no evidence of a cavity. A few bronchitic râles were scattered over the chest on both sides. The breath-sounds below the left apex were not quite satisfactory. Shortly after this she ceased to attend, and was lost sight of.

This case would appear to have been one of solitary *caseous abscess* at the right apex, the contents of which were expertorated, cicatrisation of the resulting cavity then ensuing, in a manner analogous to that sometimes seen in suppurating glands of the neck. It may be urged that as tubercle bacilli were not found—the date of the observation precluding this—the case must remain a doubtful one; but its subsequent course supports the diagnosis, and we have since observed a very similar case in private, in which bacilli were discovered, and which we will now very briefly relate.

A gentleman, aged about twenty-five years, was at a French watering-place, and on the night of his departure was dancing to a late hour, and only allowed himself time to rush in evening clothes to the boat of departure for London. He was much heated, and became severely chilled on the voyage. The next day he was seized with rigor and very severe pain in the left upper scapular region. He presented the signs of acute pneumonia, but when seen at Maidenhead in consultation with Dr. Moore within a few days of his attack, the pain in the shoulder continued, and there were notable cavernous breath-sounds and gurgling over the spinous region of the left scapula. The temperature was high and fluctuating, with marked night-sweating, and a considerable amount of muco-purulent expectoration of a very fœtid odour was being discharged. In a word, the signs and symptoms were those of an abscess in the upper and back portion of the left lung. The peculiarity of site and the general aspect of the case led to a careful examination of the sputum, which revealed the presence of tubercle bacilli in abundance. Nevertheless, to be brief, this gentleman made a fairly rapid and complete recovery, and, some fifteen years later, had married, and was in excellent health, having since engaged in big-game shooting in Africa, and having effected an insurance on his life at ordinary rates.

The case is a modern analogue to the preceding one, and may be rightly described as an example of tuberculous abscess of the lung. Similar cases have also been reported by other observers, and post-mortem observation leaves little doubt that such abscesses occur more frequently than positive clinical experience would indicate, although they are rarely single and uncomplicated, as in the above instances.

It should be especially added that when a second attack occurs it often does not affect, as in the case of A. W., the part of the lung originally attacked, but some other portion of the same lung or the opposite apex.

With the softening of the pulmonary textures the expec-

toration ceases to consist of mucus. It is no longer viscid, tenacious, and more or less frothy, but contains opaque specks and purulent streaks, and, gradually becoming more purulent, each sputum is moulded in its escape through the air-passages and clothed with a thin layer of mucus to form a more or less isolated nummular mass. With the enlargement of the cavities the sputum becomes more diffluent. As the expectoration increases in abundance, it becomes also easier, and the patient describes his cough as being looser, but soon complains of the amount of expectoration.

At an early period, when the centres of excavation are as yet minute, a careful examination of the sputum (p. 72) will reveal the presence of elastic tissue (Fig. 55), which has indeed almost exactly the same significance as that of the

FIG. 55.—ELASTIC TISSUE FROM PHTHISICAL SPUTUM. Woodcuts from photographs taken by the late Mr. F. Fowke. × 170 (about).

physical sign of moist crackling. Tubercle bacilli are at this stage always present, and generally in abundance.

A cavity of recognisable dimensions having been formed, it may increase indefinitely by solution of fresh tissue and by the coalescence with it of smaller cavities; or it may cease to extend. In the latter case it may continue to secrete much purulent fluid for a long time, or it may become quiescent and undergo more or less contraction. The ordinary method of extension and enlargement of cavities by softening down of fresh pulmonary tissue into the original cavity, and by the merging of adjacent smaller excavations into one larger one, requires no further comment. It is obvious that all trabeculated cavities have been formed in this way, the trabeculæ being the remnants of the septa which formerly partitioned the smaller cavities from one another.

There is, however, another theory, advanced by the late Professor Rindfleisch,[2] to explain the enlargement of bronchial and other cavities, which we must notice here, although there are many and important objections to its acceptance. According to this theory, the obstruction of numerous small bronchi by the pulmonary consolidations necessitates during inspiration an increase of the air-pressure upon the interior of the bronchi in front of the obstruction, and also upon the interior of cavities, thus leading to their dilatation.

Professor Rindfleisch conceived that the soft walls of recent cavities readily yield before this increased air-pressure, and thus enlarge towards the pleural surface, condensing the tissue around them. When we consider, however, that the influx of air into the lungs does not take place in any constant quantity, but awaits the aspiration dependent upon the expansion of the thoracic cavity, we see that this theory cannot apply to the enlargement of phthisical cavities nor even to ordinary cases of bronchiectasis. For in both these morbid states, but especially in phthisis, the lung is more or less consolidated or thickened, and having its pleural surfaces adherent, at least over those portions which are excavated, resists expansion more than in health. In vigorous people with healthy lungs the utmost available inspiratory force only exceeds by from two to three inches of mercury that necessary to expand the lung. In phthisis this reserve force is much diminished, and the vital capacity of the chest much lessened, so that we have less air entering the lungs and at less pressure. Hence it would seem that the inspiratory force, effective in producing certain forms of emphysema, can have but little appreciable action in dilating pulmonary cavities. We have not observed in phthisis, except in advanced cases with marked dyspnœa, any excessive effort with inspiration; the muscles of inspiration, indeed, lessen in vigour with the general wasting. During cough, however, the intercostal spaces over a superficial cavity become noticeably bulged, and with the stethoscope we may hear the air forcibly rushing into the cavity, so that doubtless the repeated cough has a tendency to dilate cavities somewhat, though even this is only an auxiliary force in effecting their enlargement.

The temperature chart of a case of phthisis with recently formed and extended cavities depicts, as we have seen, a

markedly hectic type of fever (Fig. 46, p. 478). The temperature mounts up to a considerable height, from 101° to 103° in the course of some hours during the day, the maximum being usually attained at some period between 2 p.m. and 10 p.m. From this point a fall ensues to below the normal, the subnormal curve culminating usually towards the early hours of the morning. The range of temperature is sufficiently indicated for clinical purposes by observations taken two or three times a day, provided we are careful to note the period of the day at which the fever is highest, and to record a daily observation at that time. We must be further careful to remember that a normal morning temperature means, in these febrile cases, a subnormal early morning temperature, as this has an important bearing upon treatment.

In a certain number of cases cavities, and especially those having their origin in caseous abscesses, *cicatrise* and become obliterated. Laennec described this as the natural mode of healing, and seemed to regard phthisis as incurable in the first stage of the disease. Taking the term cavity in its strict sense as meaning any loss of pulmonary substance, however small, cicatrisation is probably common; nay, we find post-mortem evidence of the cicatrisation of cavities large enough to come within clinical recognition, but they may be surrounded by other cavities and disease centres, which continue to enlarge and render cicatrisation of one amongst them of little avail. We have, however, alluded to a case in which a caseous cavity formed and cicatrised under observation, the patient remaining well for more than fifteen years afterwards.

2. **Quiescent Cavity.**—It more commonly happens that a cavity, having attained certain dimensions, becomes quiescent, all progress of the pulmonary disease being arrested; its walls then condense and toughen by the development of fibrous tissue, so as to shut it off from the surrounding lung. The contents of the cavity become less and less abundant, the sounds yielded to auscultation more and more dry, until no moist sounds at all are heard even on deep breathing, but only a few clicks and a characteristic succussion of air when the patient coughs. This kind of cavity at once begins to shrink somewhat in size, its walls becoming denser and thicker by a cicatricial process (Plate XXX.); the corresponding chest

PLATE XXX

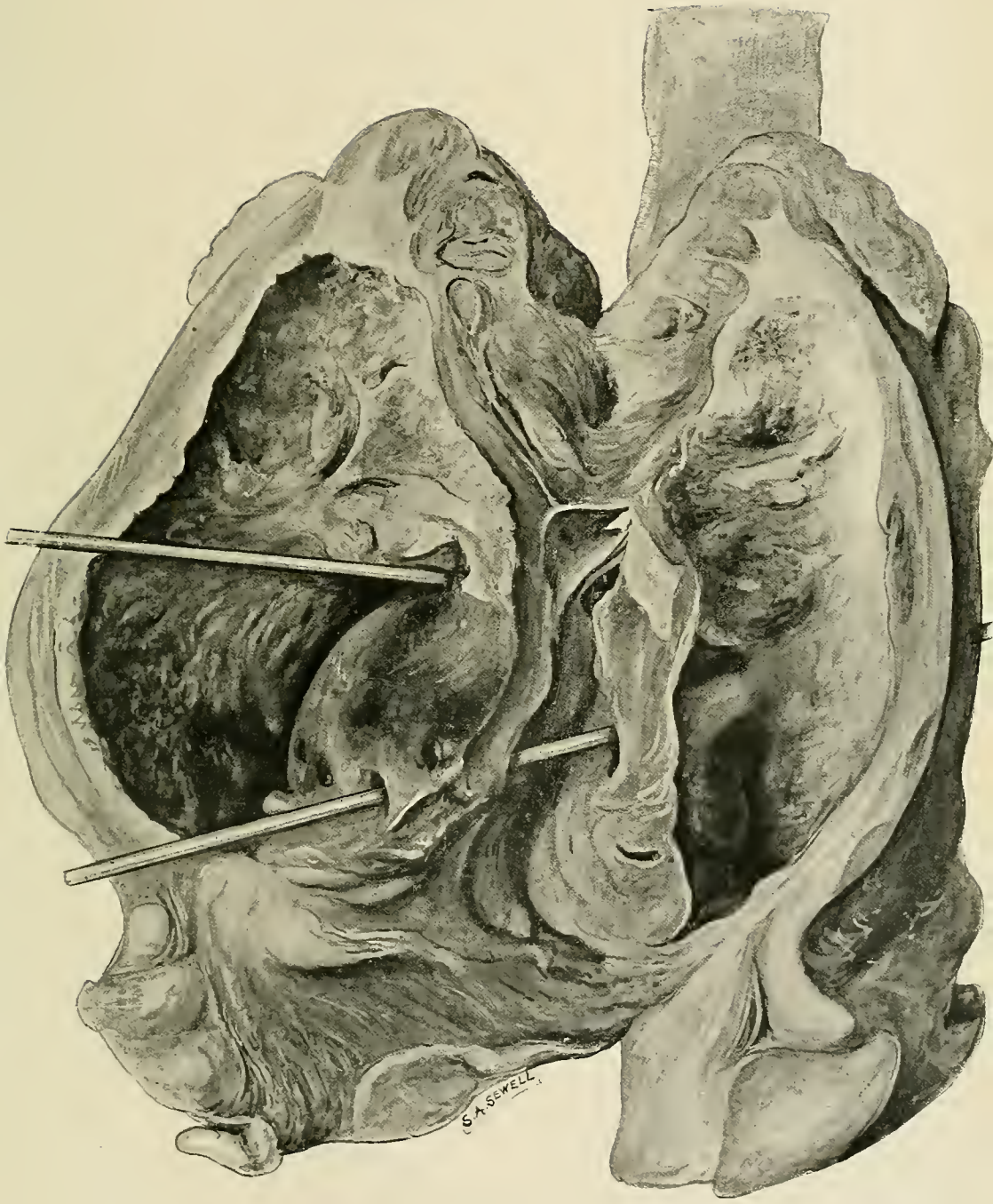
ARRESTED TUBERCULOSIS WITH TOTAL EXCAVATION OF LUNG

The drawing shows a vertical section through the left lung, the parts being then opened out. The whole of this lung is now represented by the cavity seen, which measured three inches in length, two inches in breadth. Into this open the two main divisions of the left bronchus, the positions of which are indicated by the glass rods. The cavity had greatly shrunken and become "quiescent." The pleura over it is dense and much thickened.

From a boy aged eighteen, who was admitted into the Brompton Hospital suffering from acute bronchitis, and who died of the same four days later. The left side of his chest was greatly fallen in, and the heart was much drawn over to the left. The right lung was enlarged, and showed evidence of former tuberculous disease. At the autopsy evidence of acute bronchitis was found, but none of active tuberculosis.

(From the Brompton Hospital Museum. Actual size.)

PLATE XXX



ARRESTED TUBERCULOSIS WITH TOTAL EXCAVATION OF LUNG.

To face p. 528.

wall flattens, and the heart and opposite lung encroach towards the affected side, to make up for the loss of space. If the surrounding lung be tolerably sound it will become expanded around the cavity, so that the latter, if only of moderate size and not very superficial, may become altogether obscured. It is very common post-mortem to see a longitudinal fold or wrinkle upon the surface of the lung bounded by expanded vesicular tissue, and on making a vertical section through such a wrinkle, we cut across a more or less deeply seated cavity which has evidently undergone contraction or possibly cicatrisation. Even superficial cavities may become in this way lost to clinical observation. There are in the Brompton Hospital Museum some examples of such a condition, which is well but rudely depicted in Laennec's work.[3]

That even large cavities may cicatrise and become permanently obliterated is a fact ascertained, as we have seen both by clinical and post-mortem observation, although its occurrence is no doubt rare. It is, however, quite common for the physical signs of a cavity which has undergone a certain degree of contraction to disappear, and be replaced by simply suppressed or very feeble (conducted) breath-sound. This does not necessarily arise from the cavity becoming obliterated, but from the bronchus with which it communicates becoming narrowed or occluded by the dense cicatricial growth in the cavity wall, in which the sheath of the bronchus partakes. Such a cavity, although it may, perchance, communicate with a few collateral minor bronchial tubes, is practically or completely *closed*, and this is the next best thing to its being obliterated. It diminishes in size, and ceases to take any further part in the production of pulmonary symptoms.

In this period of cicatrisation tubercle bacilli are present in greatly diminished numbers, or may be altogether absent, unless there be other excavations or tuberculous lesions present which are less quiescent.

We have observed clinically the complete loss of all signs over a cavity of considerable size, and their return after a few days, showing that a temporary closure of the bronchus had taken place, probably from a plug of mucus. The periodically abundant and fœtid expectoration found in some cases in which there can be discovered no signs of excavation is due to the cavity only communicating obliquely with a bronchus,

so that the secretion becomes pent up for some time before it can find an exit.

3. **Secreting Cavity.**—This is usually a cavity of tolerably old date which has ceased to extend, and is unaccompanied by active pulmonary disease. It is dense-walled, and is lined by a smooth opaque pyogenic (false) membrane, which can be readily scraped off, exposing a highly vascular, dusky red, subjacent surface. The trabeculæ, which are numerous, present the same vascular surfaces and false membranes. Such cavities may go on indefinitely secreting a diffluent creamy pus; they yield gurgling sounds, with marked amphoric breathing, and dull tubular percussion note. There is either no fever, or it is trivial, consisting of a slight rise of temperature only at night. The tongue is clean, with a tendency to redness and loss of epithelium. Although the appetite usually continues good, the patient slowly loses ground, and acquires the sharp hungry features peculiar to chronic phthisis, with clubbing of the fingers and toes, and a tendency to lardaceous degeneration of organs. Diarrhœa is apt to supervene, and troublesome sickness is sometimes occasioned by the cough. These cases are serious, but are less common now than formerly.

In favourable cases the secretion dries up and the cavity becomes quiescent. Unfavourable cases gradually fail from lardaceous disease of other organs or from recurrent diarrhœa. Also there is, in these cases especially, the danger of some of the abundant secretion becoming inhaled during cough into portions of the same or of the opposite lung as yet unaffected, and thus setting up fresh centres of disease.

Hæmoptysis is not common from either the quiescent or the secreting cavity, but it sometimes occurs in a dangerous and unexpected manner from the rupture of an ectasia or aneurismal dilatation projecting from the unsupported cavity side of a large pulmonary vessel.

4. **Ulcerous Cavity.**—This last kind or condition of cavity, which is also more rarely met with in these days of aseptic treatment, originates in the usual way, and may have been quiescent or merely secreting for some time, when, from exposure to septic influences, or from other causes, it assumes a state of active ulcerative extension. We have known such cases to be endemic in a ward which was overcrowded, and to have ceased on a bed being removed. It cannot, indeed, be

too carefully remembered in the treatment of tuberculous affections of the lungs that all such patients have internal *wounds* or *sores*, which, unlike most other internal affections, are accessible to the contamination of foul air, and that septic processes may be readily set up in them, which are apt to be recognised only as "*intercurrent pneumonias*" or other local inflammations.

Ulcerous cavities are angry-looking, deep dusky red on their inner surfaces, often studded with hæmorrhagic points or ulcerative erosions; they are highly trabeculated and very irregular in shape, but sharply demarcated from the lung tissue by a thin vascular wall. They contain a copious blood-stained purulent secretion, which, when expectorated, is mixed with the ropy mucus from the intensely vascular bronchi which communicate with them. The lung tissue surrounding the cavity is engorged and œdematous, and at distant parts of the lungs pneumonic centres may be found, which evidently owe their origin to the inhalation of the acrid secretions from the cavity. In such cases sharp fever is present, with quick pulse, furred tongue, and a tendency to typhoid symptoms. The expectoration is usually mixed with blood, or dark altered clots may be removed from the cavity. Sometimes copious hæmorrhage takes place from the erosion of a large vessel.

The prognosis in cases of ulcerous cavity is always grave and hazardous, but if it be borne in mind that the aggravation of symptoms is to be explained rather by insanitary conditions surrounding the patient than by an accession of genuine tuberculous activity—in other words, that the condition is septic rather than tuberculous—an appropriate measure of treatment corresponding to this view will often considerably amend the prospects of the patient.

The treatment of these conditions will be dealt with in a later chapter (p. 688).

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CHAPTER XXXVII

THE COMPLICATIONS OF PULMONARY TUBERCULOSIS

Laryngeal, Aural, and Intestinal Tuberculosis.

THE complications met with in the course of tuberculosis of the lungs are many and varied. The relative frequency of the most important, as deduced from the autopsies of 263 consecutive cases (188 males, 75 females) of chronic pulmonary tuberculosis, in which the post-mortem examinations were made by one of us, is shown in the following table:

TABLE SHOWING RELATIVE FREQUENCY OF THE MORE IMPORTANT COMPLICATIONS OF CHRONIC PULMONARY TUBERCULOSIS, BASED UPON 263 CONSECUTIVE AUTOPSIES (188 MALES, 75 FEMALES).

	Males.	Females.	Total.
	Per Cent.	Per Cent	Per Cent.
Larynx—tuberculous ulceration of ...	54·7	46·9	52·6
Trachea— ,, ,, ...	27·1	20·0	25·1
Main bronchi— ,, ,, ...	12·2	10·6	11·4
Pneumothorax	7·4	4·0	6·4
Pleurisy—(1) acute sero-fibrinous	6·3	—	4·5
(2) suppurative (empyema) ...	2·1	2·6	2·2
Pulmonary aneurism	10·6	5·3	9·1
Fatal hæmoptysis	9·0	4·0	7·6
Pericarditis—tuberculous	2·1	—	1·5
Veins—thrombosis of	1·0	6·6	2·6
Peritonitis—tuberculous	3·7	2·6	3·4
Intestines—tuberculous ulceration of ...	61·3	61·3	61·3
Fistula *in ano*	1·6	—	1·1
Lardaceous disease of viscera	9·5	13·3	10·6
Kidneys—(1) miliary tuberculosis of ...	11·1	9·3	10·6
(2) ulcerative tuberculosis of ...	2·6	1·3	2·3
Suprarenals—tuberculous disease of ...	2·1	2·6	2·3
Bladder—tuberculous cystitis	1·0	—	0·7
Male generative organs — tuberculous disease of	4·7	—	—
affecting (1) body of testicle	1·0	—	—
(2) epididymus	3·2	—	—
(3) vesiculæ seminales ...	2·6	—	—
(4) prostate	3·2	—	—
Female generative organs—tuberculous disease of	—	10·7	—
affecting (1) ovaries	—	2·7	—
(2) Fallopian tubes	—	10·7	—
(3) endometrium	—	6·7	—
Meningitis—tuberculous	2·6	2·6	2·6

Let us now consider some of the above complications in more detail.

Laryngeal Tuberculosis.

It sometimes happens in the adult that pulmonary tuberculosis is ushered in with laryngeal symptoms, and the variety of the disease thus arising ranks amongst the most fatal and distressing of all.

Strictly speaking, the term "laryngeal phthisis" should be applied only to those very rare cases in which the tuberculous lesion of the larynx is primary; but in practice it is more loosely employed to include all those cases in which laryngeal symptoms constitute an early and striking feature. This latter employment of the term is not only more convenient, but is also more in accordance with the general pathology of the disease; for although in many instances it is the affection of the larynx which first attracts attention, yet even in these cases the lungs speedily show obvious involvement, so that to attempt more rigidly to restrict the term would imply, what is not the fact, that there is a phthisis which begins and ends with laryngeal disease.

In the analysis of our consecutive autopsies upon cases of chronic pulmonary tuberculosis,[1a] in 247 of which the larynx was examined, we found naked-eye evidence of involvement of the larynx in 130, or 52·6 per cent.; in 89 cases, or 36·0 per cent., the ulceration was "extensive." Probably the latter figures represent more nearly the percentage of cases in which symptoms referable to the larynx are complained of during life. The sexes would appear to be affected equally, but it is remarkable how exceedingly uncommon the complication is in children.

The following table, based upon our 247 autopsies, shows certain further points of interest in regard to the condition:

TABLE SHOWING THE LESIONS MET WITH IN THE LARYNX AFTER DEATH IN 247 CASES OF CHRONIC PULMONARY TUBERCULOSIS.

	Number of Cases Affected.	Percentage
Larnyx—Tuberculous ulceration, total cases ...	130	52·6
Slight ulceration	41	16·6
Extensive ulceration	89	36·0
,, laying bare left arytenoid ...	30	12·1
,, ,, ,, right arytenoid	20	8·1
,, ,, ,, the thyroid ...	1	0·4
,, destroying upper portion of epiglottis	18	7·0
Œdema of glottis	3	1·2

Tuberculous disease of the larynx most commonly arises as a complication in advanced pulmonary phthisis. It is, however, by no means restricted to such cases, and it has been shown at the King Edward VII. Sanatorium, Midhurst, by Sir StClair Thomson,[2] that even in the early cases included in Group I. 13·7 per cent. showed evidence of laryngeal involvement, the percentage rising to 27·1 among the patients in Group II., and to 40·8 in Group III.

The way in which the larynx becomes affected has been much debated, but there can be little doubt that it is the result of direct contact of the sputum with the mucous membrane, the expectoration tending to cling to any crannies and crevices that it meets with in its passage outwards. The bacilli possibly sometimes enter the laryngeal tissue through superficial erosions, or pass through the unbroken mucous membrane, as in the case of the intestinal wall. But Dr. Jobson Horne[3] has demonstrated that they may enter through another portal, the ducts of the muciparous glands of the submucosa; and very probably this is the most common mode of entrance, since, as he points out, the parts of the larynx richest in glandular structures, such as the arytenoid eminences, and especially the interarytenoid region, are also those most vulnerable to tuberculous infection.

The bacilli having gained access to the larynx, the progress of the disease is similar to that in other organs. Tubercles form in the submucosa, producing tumefaction and œdema. Later, caseation and softening occur, with the production of minute ulcers, which, by their coalescence, form larger ones, the floors and walls of which are the seat of fresh tuberculous deposits.

The morbid appearances seen with the laryngoscope during life vary according to the period of the disease. In the early stage a marked and general anæmia of the laryngeal mucous membrane is often present, which, if it does not form part of a general anæmia, should be regarded as very suggestive of tubercle. In other cases the larynx is hyperæmic. Later, as the tubercles enlarge, infiltration and œdema become visible to the naked eye. The positions in which these appearances are first seen differ. Our own experience would lead us to place the first signs in the interarytenoid space or in the aryepiglottic folds, and this is in accordance with the experi-

ence of Dr. Horne,[4] who, in a careful analysis of 359 consecutive cases of pulmonary tuberculosis, found that the parts were affected in the following order:

Interarytenoid space	in 176	cases.
Arytenoid eminences (bilaterally)	,, 139	,,
Ventricular bands (bilaterally)	,, 57	,,
Epiglottis (free edge)	,, 30	,,

The early changes which thus present themselves in the interarytenoid space, and which are so important from a diagnostic point of view, are perhaps hardly as yet sufficiently appreciated. They may consist merely of a slightly irregular swelling of the mucous membrane, resulting from the deposition of tubercle beneath; but in the majority of cases the swelling is more marked, and in a few, definite tuberculous *tumours* will be found, pale and with irregular edges, which constitute perhaps the only evidence of laryngeal tuberculosis.

As the œdema increases, characteristic changes may be produced, the swelling over the arytenoids and in the ary-epiglottic folds presenting the well-known "pear-shaped" appearance, while the epiglottis may become pale and swollen, or, as it has been termed, "turban-shaped."

Ulceration later supervenes, the favourite seat being over the glottic aspect of the arytenoid cartilages, where a ragged ulcerous hole is often found, at the bottom of which after death the denuded and roughened cartilage may sometimes be felt. The interarytenoid space, the ventricular bands, the vocal cords and epiglottis are also frequently the seat of ulceration, and we have seen instances in which ulceration has extended deeply from the anterior junction of the vocal cords to produce perichondritis of the thyroid cartilage with involvement of the subcutaneous cellular tissue. Ulceration in the more deeply seated parts may or may not be observed by the laryngoscope, but intense injection of the mucous membrane is often visible. In advanced cases the orifice of the glottis may be seen distorted by ulceration and thickening, the cords being involved in the havoc so as to render their approximation impossible, and the arytenoid articulations on one or both sides participating in the disease. In this stage, purulent secretion collects in the ventricles and about the cords.

With severe ulceration of the larynx, it is common to find a similar condition of the trachea, and less often of the

bronchi. In the trachea the ulceration tends to spread from minute points, and gives a peculiar moth-fretted appearance to the surfaces. In other cases the ulcers are larger, and frequently surrounded by a brilliant red zone of injection; eventually the tracheal cartilages are often laid bare.

Symptoms.—The sufferings of the victim of larnygeal phthisis are great and varied. One of the first symptoms that attracts attention is an alteration in the tone and quality of the voice. It becomes husky and usually deepened. Vocalisation is uncertain, the voice sometimes falling into a husky whisper, to reappear with deep, reverberating tone when an increased effort is made in speaking. In some cases, however, even at the earliest stage, the voice is suppressed from an inability to approximate the cords. This may be due to general loss of muscular and nervous tone, or may result, as Dr. Horne has shown, from an inflammation of the laryngeal muscles, a myositis, which impedes their functional activity. In less early stages, again, it may possibly be mechanical, the result of the presence of a tuberculoma in the interarytenoid space, such as we have described, or in rare cases arising from compression of one or other recurrent laryngeal nerve, the right, as a rule, from apical pleurisy, the left by enlarged and probably tuberculous glands.

If the laryngeal disease be only slight, and the patient's lung trouble be quiescent or show signs of healing, there may be no other symptoms, and, with suitable treatment, including absolute rest to the voice, the laryngeal as well as the pulmonary disease may become arrested. Sir StClair Thomson[2] estimated at the King Edward VII. Sanatorium that arrest of the laryngeal disease was obtained in one-fifth (20·7 per cent.) of the cases. But, encouraging though this is there can be no question that the prognosis in phthisis, at whatever stage of the disease, is rendered much more grave by the presence of the complication, the mortality among such patients being considerably greater than in the case of those whose larynx remains unaffected.

As the disease in chest and larynx progresses, the patient's condition becomes a pitiable one. He loses flesh, suffers from sweats and evening fever, and experiences oppression of breathing, which may amount to serious dyspnœa, both respiration and pulse being quickened. A troublesome teas-

ing cough of a harsh, dry character is complained of, which is attended with pain and a sense of rawness in the throat, and with scanty and difficult expectoration. Pain on deglutition is a frequent symptom, and it is still more characteristic when described as shooting upwards to the ears, causing in them "pricking sensations." On deep pressure in the upper tracheal region some tenderness is elicited. As the disease proceeds and the ulcerative destruction of the larynx extends, the aphonia becomes complete, and the cough most distressing and paroxysmal. At this period the lung disease has usually proceeded to excavation, and the expectoration is more abundant; effectual cough is, however, almost impossible, in consequence of the patient being no longer able to bring about its essential condition, closure of the glottis. Deglutition is now difficult and painful, from the pharyngeal muscles compressing the tender and swollen parts, and from the irritable, stiffened, and often ulcerated epiglottis failing effectually to guard the laryngeal aperture.

The fauces and tonsils may be normal in appearance, but they are often pale, and drier than natural, with their small subcapillary veins too well marked. The back of the pharynx is frequently granular, even abraded, with streaks of viscid mucus adherent to the surface. The mucous glands are too prominent. A distinct ulcer, having the characteristic raised irregular margin and granular ash-coloured surface of the tuberculous ulcer, may sometimes be seen at the back of the pharynx or behind one of the tonsils.

In rare cases an ulcer appears upon the tongue, or on the inside of the cheek, especially about the orifice of the parotid duct. Such cases may suggest syphilis, but are by no means necessarily of this nature, although careful inquiry on this point should always be made.

Diagnosis.—In well-marked cases of tuberculous laryngitis the diagnosis is not difficult. The diseases which most simulate it are chronic alcoholic or irritative catarrh, especially that variety produced by excessive cigarette-smoking, syphilitic disease, and hysterical aphonia. More rarely it has been mistaken for malignant growth.

With regard to the first-named—the chronic catarrh arising from drink, dust, or smoking—the absence of fever, the presence of a definite exciting cause, and the laryngoscopic

signs of general catarrh without local thickening or ulceration, are usually sufficient to distinguish it from laryngeal tuberculosis. Alcoholic and smoker's catarrh are, moreover, always associated with a similar affection of the pharynx. In the absence, however, of a definite exciting cause, a catarrh which does not speedily clear up under appropriate treatment should always be viewed with suspicion. In any doubtful case the sputum must be examined; tubercle bacilli are invariably present in the tenacious, glairy mucus expelled in cases of tuberculous laryngitis which have proceeded to ulceration.

In syphilis, during the secondary stage, the larynx may present a condition of catarrh, but the general symptoms of the disease should in this case guide the diagnosis aright. In the tertiary stage gummatous swellings, either localised or diffused, may make their appearance. They are usually deep red in colour, and soon proceed to ulceration. Such ulcers are deeper and more sharply cut than those occurring in phthisis; they are also much less painful. Gummata may occur in any portion of the larynx, whether in the region of the arytenoids, the true or false vocal cords, or elsewhere; but a favourite seat is the epiglottis, leading to the extensive destruction which is so suggestive of syphilis, although it may occur as the result of tubercle. Much contortion or deformity from cicatricial change should also lead to a suspicion of syphilitic disease. A rapid improvement under a course of iodide of potassium or salvarsan will assist the diagnosis.

Passing to functional aphonia, it not infrequently happens in early phthisis, as we have already indicated, that the voice from time to time is weak and whispering from inefficient adduction of the cords, and the patient shows all the symptoms of a functional loss of voice. The larynx may be anæmic, but often presents no other sign of tuberculosis, although it is probable that in many cases tubercles have already formed. Whenever a patient, therefore, presents symptoms of functional aphonia, the possibility of tuberculosis should be borne in mind, especially if the aspect or family history renders such a disease likely. The lungs must be examined with great care, and the weight and temperature chart closely observed.

It has sometimes happened that the tuberculous tumours to which we have referred, and which are not by any means limited to the interarytenoid region, have been mistaken for

malignant growths, and operated upon accordingly. In such cases, if, after careful examination of the lungs and of the sputum, as well as of the appearances of the larynx and the situation of the tumour, doubt still remains, a portion of the mass must be removed for microscopical examination. Another condition which may be sometimes mistaken for laryngeal tubercle is a warty excrescence from one or other cord, a condition causing partial aphonia and one not uncommonly associated with cough and pulmonary catarrh. Laryngeal examination and removal of the growth will settle the diagnosis and cure the symptoms.

The treatment of laryngeal tuberculosis will be considered in a later chapter (p. 697).

Tuberculous Disease of the Middle Ear.

This manifestation of tuberculosis, though not uncommon in infants under two years of age, is rare as a complication of phthisis in the adult, though we have met more than one instance of it. When occurring in connection with phthisis it manifests itself by deafness in the affected ear, accompanied by discharge, but *without pain.* It is thus distinguished from the more common septic variety of acute otitis media, in which earache is at the commencement a prominent feature. On examination the tympanic membrane is seen to be reddened, swollen and thickened, an index of a similar condition of the mucosa lining the tympanic cavity, and a perforation is often visible. The discharge is apt to be watery and curdy, and tubercle bacilli may be found in it.

The prognosis as regards hearing in the affected ear is poor, but if the pulmonary tuberculosis becomes quiescent and the patient's general health remains satisfactory, the lesion in the ear will probably undergo arrest and the discharge cease.

Treatment consists in the employment of sanatorium and other methods calculated to benefit the lung disease and to raise the vitality of the patient. The ear should be carefully cleansed by the daily use of peroxide of hydrogen, Except when especially exposed to dust, as in motoring, the meatus should not be closed with cotton-wool, the fresh air having a beneficial action on the lesion in the ear, just as it has upon the lungs.[5]

Intestinal Tuberculosis.

Ulceration of the bowels is sometimes met with as a primary disease in young children. It is a very common complication of phthisis—indeed, it may be said to be one of the attendant lesions of the disease. Of the 263 consecutive cases alluded to in our table (p. 532) it was present in 61·3 per cent., both sexes being affected equally.

Excluding the appendix, intestinal tuberculosis is scarcely ever met with in adults except in association with tuberculous disease of the lungs, and is commonly due to the swallowing of the infected sputum. It most usually complicates the later stages of the disease, but it may, like laryngitis, occur at an early period, at a time when pulmonary physical signs—always somewhat masked during diarrhœa—are difficult to detect. Anything that tends to derange the digestion and the bowels favours the occurrence of the complication. Tubercle bacilli are present in the intestinal lesions, and may be discovered in the evacuations.

The disease commences with inflammatory swelling of certain of the follicles of the small or large intestine. Caseation follows, and the softened products discharge into the intestine, leaving an ulcerous recess behind. Both in sections of the ulcerated tissues and in the discharges from their surfaces tubercle bacilli are found.

On the peritoneal surface of the bowel, over the site of an ulcer thus established, flakes of lymph are often to be seen, and inflammatory adhesions may be formed with an adjacent coil of intestine. The outline of the ulcer can be distinguished shining through the peritoneal surface, and over its base granulations of tubercle are to be observed. These granulations are connected with the lymphatic vessels, some of which form white lines or streaks over and about the site of the ulcer. The ulcer is at first circular in shape, and soon comes to have the characteristic thickened edge and irregular warty base which distinguish it from the typhoid ulcer. It tends to enlarge in a transverse direction, beyond the limits of the gland follicle, in which it originated, the extension following the direction of the vessels, and being determined by the formation of tubercles in their sheaths.

Whether the ulcers be single or in groups depends upon the

glands, whether solitary or agminate, which are attacked. Peyer's patches are favourite seats of ulceration. In them irregular serpentine ulceration arises by extension and coalescence from several centres, often leaving small tracts or islets of mucous membrane intact. All the gland follicles of a patch are by no means necessarily affected; the ulceration may only involve a certain number of them, extending transversely to the mucous membrane beyond. In the cæcum, too, the ulceration is often very extensive, the whole of the mucous membrane being eroded for several inches, with the exception of small islets or streaks here and there, which have escaped and stand out prominently, indicating the original extension of the ulcerative process from many centres. More or less general hyperæmia of the mucous membrane, very variable in amount at different times and in different places, attends the ulcerative process. Considerable general thickening of the large intestine is sometimes also present.

As already mentioned, local peritonitis commonly attends the intestinal lesion, and adhesions are frequently formed. Perforation of the bowel occasionally occurs, sometimes leading to the escape of fæcal matter and to general peritonitis, sometimes to the establishment of a fistulous communication between adherent portions of intestine, and, again, in other cases producing collections of pus localised by surrounding adhesions. These processes are strictly analogous with those which occur in connection with the pleural surface of the lungs in phthisis. Drs. W. S. Fenwick and Dodwell,[6] from inspection of the records of the Brompton Hospital, found 25 instances out of 2,000 cases of phthisis (1·2 per cent.) in which perforation occurred from this cause. Our own post-mortem observations[1b] gave a very similar result—viz., 3 cases out of 263 autopsies, or a percentage of 1·1, the perforation in each of these instances occurring in the ileum. We may add that we have three times known general septic peritonitis to result from deep tuberculous ulceration of the bowel, but without perforation.

The lower two or three feet of the ileum and the cæcum are almost invariably the portions of bowel involved in tuberculous disease, but the ulceration may extend both above and beyond these points to the duodenum and the rectum. The appendix also is frequently affected, and tuberculous appen-

dicitis is not of uncommon occurrence as a primary malady. The following table shows the relative frequency with which the different portions of the bowel were attacked in our series of autopsies, in 61·3 per cent. of which tuberculous ulceration of the bowel was present: [1c]

TABLE SHOWING THE RELATIVE FREQUENCY WITH WHICH TUBERCULOUS ULCERATION WAS FOUND IN DIFFERENT PORTIONS OF THE BOWEL IN 263 CASES OF CHRONIC PULMONARY TUBERCULOSIS.

	Number of Cases.	Percentage.
Duodenum	10	3·8
Jejunum	53	20·3
Ileum	125	47·8
Cæcum	114	43·6
Appendix	98	37·5
Colon	83	31·8
Rectum	35	13·4
Fistula *in ano*	3	1·1

It will be seen that the ileum, cæcum, and appendix are the favourite seats of ulceration. We may add that in 109 cases, or 41·7 per cent., ulcers were found in both the small and large intestine; in 19, or 7·2 per cent., they were restricted to the small intestine; in 24, or 9·2 per cent., to the large; and in 8, or 3 per cent., they were found only in the appendix.

Symptoms.—The usual symptoms of ulceration of the intestines are diarrhœa and pain in the abdomen. There is nothing at first which can be noted as peculiar in the character of the diarrhœa; the stools are pale and loose, resembling those of ordinary intestinal catarrh, and occasioned doubtless by the presence of such catarrh. There may be some nausea; the tongue is furred, with red tip and edges and prominent papillæ, and the patient complains of thirst. The pain is usually referred to the umbilical region; it is of a colicky nature, and no marked tenderness is present over any special area of the abdomen. There is notable irritability of the whole mucous tract, and the taking of food, and more often warm drinks, into the stomach causes the bowels soon to act. The looseness is, however, at this stage tolerably amenable to treatment, and for a time the motions become natural, or the patient is even constipated. Soon, however, a relapse takes place, and the diarrhœa is more obstinate than before. Now some decided tenderness may be felt on deep palpation, most likely in the right iliac region; the motions become more scanty,

some mucus is passed with them, a speck or two of blood may be observed, or a teaspoonful or more may escape. Anything approaching to copious hæmorrhage is, however, rare. The tongue becomes red and patchy from loss of epithelium; it may present short transverse fissures on each side of the median line.

The further symptoms vary with the seat of the principal ulceration. If this be limited to the ileum, the diarrhœa may for a long time be held in check by treatment; but the bowels are irritable, the abdomen somewhat prominent in the umbilical region, and tender. If the ulceration, on the other hand, has its principal seat in the cæcum, the tenderness over that region is more marked, the diarrhœa is difficult to control, blood and mucus are frequently present in the stools, and the patient acquires a pinched look and rapidly loses flesh. Ulceration extending farther down the colon to the rectum is signified by more distinctly dysenteric symptoms, pain and tenderness over the arch of the colon, more frequent mucous stools with tenesmus; the gastric symptoms, on the other hand, are less marked, and considerable appetite, with a fairly clean tongue, is often retained.

It is easy to note down these symptoms, as they occur in case after case of this dreaded complication; but it is much more difficult to fix upon any symptom, if any, indeed, exist, that is positively characteristic of ulceration. The plain rule is, in patients with tuberculosis, always to treat intercurrent diarrhœa as though it were due to commencing ulceration of the intestines.

But in some cases the diarrhœa precedes, or at the time of observation altogether masks, the lung symptoms; and it is remarkable how completely even decided pulmonary disease may be thus obscured. The cough and expectoration may cease or become trivial, and the dryness of the pulmonary tissue gives an exaggerated "vesicularity" to the respiratory murmur that masks existing defect. The recognition of this fact is of much importance in diagnosis, the alternation in prominence between the chest and abdominal symptoms being characteristic of intestinal tubercle.

It is unnecessary to dwell upon the later stages of this disease: rapid wasting and exhaustion from the constant uncontrollable diarrhœa, a depression of the previously some-

what raised temperature, aphthous mouth, and lividity of extremities, are the closing symptoms.

It is a fact worthy of note that even extensive ulceration of the intestines may exist without any diarrhœa. Dr. Walshe[7] observed: "Not only may pretty extensive ulceration exist in the ileum without pain, either spontaneous or elicited by pressure, but with a confined state of bowels. Again, I have known, in a case running an acute course, marked abdominal pain and tenderness, conjoined with obstinate constipation, where, after death, the bowels—in spite, too, of the frequent use of purgatives—contained abundant solid fæces, and the ileum was extensively tuberculised and ulcerated."

In the *Pathological Transactions* for 1868, a case of chronic phthisis with extensive and deep ulceration of the ileum is recorded by one of us,[8] in which constipation was a marked symptom throughout the patient's illness; and we have met with other instances. There is no doubt that the diarrhœa is, to a large extent, dependent upon irritative catarrhal inflammation of the mucous membrane in the neighbourhood of the ulcers, and this is probably the reason that it is a more prevalent symptom in the late summer and autumn months than in the winter. In some cases, however, the ulcerations are so deep as extensively to destroy the muscular coat, and thus materially to interfere with the peristaltic movements of the intestines. In others of a more acute kind the peritoneum may be much involved, and the muscular coat paralysed. An analogous state of things in both these respects sometimes obtains in typhoid fever, in which it is not at all uncommon for constipation to be present throughout, and we have known such cases to terminate fatally by perforation.

These latter considerations bring home to us the great importance of a very careful treatment of constipation in phthisis. We have seen perforation occur from the too hasty administration of a couple of colocynth and mercurial pills for constipation, which was present together with ulceration. The stronger purgatives should, therefore, only be administered with great caution, and the symptom should, in fact, be always combated with the utmost gentleness. For a full consideration of the treatment of this important complication we must refer the reader to a later chapter (p. 694).

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(*b*) *Loc. cit.,* p. 12.

(*c*) *Loc. cit.,* p. 14.

[2] (*a*) "Three Years' Sanatorium Experience of Laryngeal Tuberculosis," by Sir StClair Thomson, M.D., F.R.C.P., F.R.C.S., *British Medical Journal,* 1914, vol. i., pp. 801, 818, and 828. See also—

(*b*) "The Prognostic Importance of Tuberculosis of the Larynx," by Sir StClair Thomson, *The Lancet,* 1919, vol. ii., p. 689.

[3] Contribution to "A Discussion on the Treatment of Laryngeal Tuberculosis," by W. Jobson Horne, M.D., *British Medical Journal,* 1905, vol. ii., p. 1188.

[4] "The Channels of Infection in Tuberculosis, and the Part played by the Lymphatic Glands in arresting, modifying, or propagating the Infection, and in preventing Recurrence of the Disease, considered with Reference to the Throat, Nose, and Ear," by Jobson Horne, M.D., *The Journal of Laryngology, Rhinology, and Otology.* London, 1907, vol. xxii., p. 281.

[5] (*a*) For a discussion on the subject of "Tuberculosis of the Auditory Apparatus," see *Proceedings of the Royal Society of Medicine, Otological Section,* 1915, vol. viii., p. 15. See also—

(*b*) "The Clinical Diagnosis and Surgical Treatment of Tuberculosis of the Temporal Bone," by W. Jobson Horne, M.D., M.R.C.P., *British Medical Journal,* 1903, vol. ii., p. 77.

[6] "Perforation of the Intestine in Phthisis," by W. Saltau Fenwick, M.D., and P. R. Dodwell, M.B., *The Lancet,* 1892, vol. ii., p. 133.

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CHAPTER XXXVIII

ON HÆMOPTYSIS

HÆMOPTYSIS or blood-spitting may be defined as the expectoration of blood from or through the lungs or bronchial tubes, and must be distinguished from *false* or *spurious* hæmoptysis, in which the blood is derived from the naso-pharyngeal or buccal mucous membrane.

The causes of hæmoptysis may be thus enumerated:

1. *Hæmorrhage from the Pulmonary Artery or its Capillaries*—

(*a*) Rupture or wound of the lung from external violence.

(*b*) Active hyperæmia of the lungs, whether inflammatory or induced by violent effort or excitement. Such active hyperæmia may be primary as regards the lungs, or may supervene or be attendant upon disease, such as tubercle, cancer, hydatid, paragonimiasis (see p. 76), etc., already present in them. In very rare cases it may be vicarious in nature.

(*c*) Mechanical hyperæmia of the lungs, secondary to heart disease, pulmonary overstrain, as in whooping-cough, embolism of one of the branches of the pulmonary artery, or pressure upon the pulmonary veins from tumours, such as enlarged glands, growths or aneurism.

(*d*) Changes in the blood, whether toxæmic, purpuric, scorbutic or otherwise, resulting in capillary hæmorrhages.

(*e*) Necrotic division of vessels in the course of softening of consolidations, such as are met with in phthisis, syphilis or cancer.

(*f*) Aneurismal dilatation, or simple erosion of branches of the pulmonary artery exposed in the course of excavation of the lung, or ulceration of the bronchial mucous membrane.

(*g*) Atheroma of the pulmonary artery within the lung.

2. *Hæmorrhage from the Bronchial Artery or Capillaries*—

(*a*) Capillary hæmorrhage from the bronchial membrane of

hyperæmic origin, or resulting from excessive blood-pressure, hæmophilia, purpura, scurvy, or toxæmic conditions.

(*b*) Ulceration or erosion of a branch of a bronchial artery.

3. *Hæmorrhage from the Aorta or One of its Great Branches.*—Aneurism rupturing through the lung or into a bronchus.

The above enumeration will suffice to remind the reader that hæmoptysis is a symptom attendant upon many morbid conditions of heart, lungs, vessels and blood. For the most part this symptom is included and sufficiently discussed in the descriptions of the diseases in which it occurs. In some cases of phthisis, however, hæmoptysis, from its profuseness, frequent recurrence, and secondary consequences, takes so important a part as to merit separate consideration. Taking out of the above list the causes of hæmoptysis operative in phthisis, they will be found to be: (1) active or inflammatory hyperæmia; (2) morbid conditions of small vessels; (3) erosion or aneurismal dilatation of larger vessels.

Active or inflammatory hyperæmia is generally present in early phthisis, and is a prominent feature in the exacerbations of the disease. Hæmorrhage is by no means necessarily attendant upon this condition, and does not usually amount to more than coloration or streaking of the sputum.

In the very earliest stage of the disease, however, as it affects successive portions of the lung, the minute vessels are softened and more or less narrowed, or even completely closed by tuberculous growth. From this combination of softened vessel wall and increased local blood-pressure rupture may ensue, or it is not impossible that it may result from minute aneurismal dilatation, after the manner described by Charcot and Bouchard in cases of cerebral hæmorrhage, but such a condition has not yet been demonstrated in the lung. The very considerable hæmoptyses in the earlier periods of phthisis, and many of the intercurrent hæmorrhages, are attributable to one or other of these morbid conditions of the small vessels, in conjunction with, or independent of, active hyperæmia. Such a hæmorrhage may be one of the first symptoms of pulmonary tuberculosis, and it was this fact which led many of the older physicians, from Hippocrates downwards, to believe in the reality of a *phthisis ab hæmoptoë*, in which the

disease was thought to originate in a bronchial or pulmonary hæmorrhage, the inhaled blood irritating the lung, and leading subsequently to the morbid changes characteristic of tuberculosis.

As the destructive changes in phthisis advance, the larger vessels of the affected portions of the lung become softened and then torn across, and although as a rule their lumen has already been obliterated by previous thrombosis, it occasionally happens that such is not the case, and more or less serious hæmorrhage ensues.

Aneurism of a branch of the pulmonary artery is a common source of fatal, and no doubt frequently also of non-fatal, hæmoptysis. The first reported case of pulmonary aneurism was, we believe, that published by Dr. Fearn[1] in *The Lancet*, February 6, 1841. Attention was later attracted to the subject by the cases of Drs. Cotton[2] and Quain,[3] published in 1866.

These aneurisms are usually situated on a pulmonary vessel which has become exposed as it crosses the wall of a cavity, the aneurism forming a projecting sac from the vessel into the cavity. Loss of support of the vessel on the cavity side, and chronic inflammatory changes in its walls, are the causes which lead to their formation. In size they vary from that of a pea to an unshelled walnut. They may be found at any age, provided the conditions suitable for their occurrence are present, but they occur most frequently in chronic quiescent cavities of old date, where the conditions for their production are most favourable (Plate XXXI.). It is to be noticed that in chronic phthisis especially (see p. 504) the general blood-pressure may remain relatively high, and clinical evidence is in favour of the probability of this being more marked on the pulmonary side.[4] Aneurisms are, however, sometimes met with in association with recent disease (Plate XXXII.), although the vessels are more apt to become occluded in such acute processes. In a series of 23 cases of fatal hæmoptysis occurring in phthisis which we have recorded,[5] in 13 the aneurisms occurred in cavities of some standing, with obvious fibrosis around; in the remaining 10 the process was a much more recent one, the surrounding lung being the seat of active tuberculous disease.

It occasionally happens that a considerable aneurism of the pulmonary artery occurs in a lung in the earliest stage of

PLATE XXXI

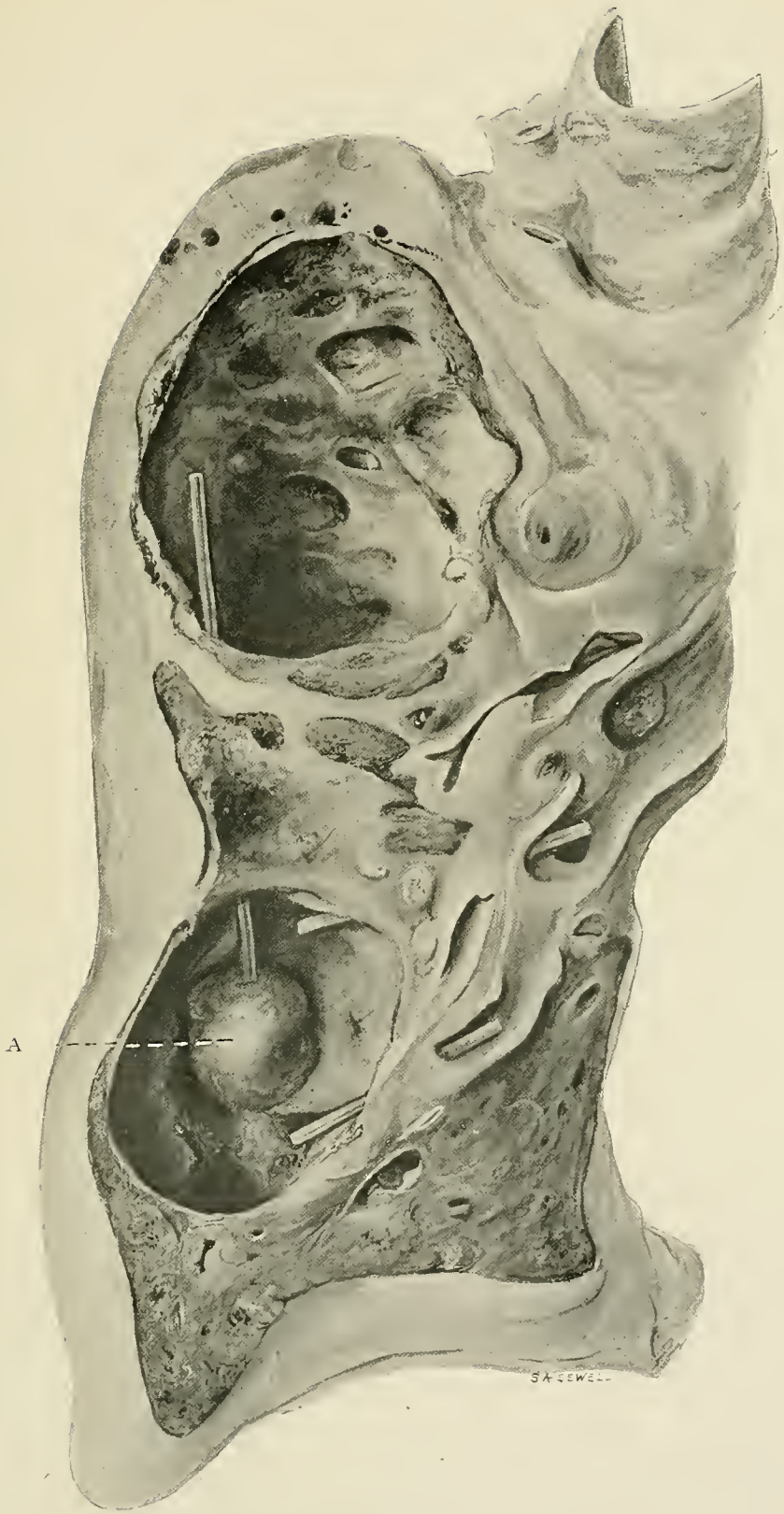
RUPTURED PULMONARY ANEURISM IN A CHRONIC CAVITY

The drawing shows a section of the left lung. The organ is shrunken, and the pleura over it dense and greatly thickened. The upper lobe is seen to be in a condition of almost total excavation, and to communicate with a large cavity in the lower lobe, on the anterior wall of which an aneurism (A), the size of a small marble, is situated. This had ruptured near its upper portion, and at the autopsy the lung was found filled with recent blood-clot.

From a man aged twenty, a shop-assistant, who suffered from chronic pulmonary tuberculosis of two years' standing. During the last week of his life he suffered on three occasions from hæmoptysis, bringing up 7, 14, and 5 ounces respectively. In the right lung a second aneurism, unruptured, but of very similar size to the one here depicted, was found (Plate XXXII.).

(From the Brompton Hospital Museum. $\frac{5}{6}$ natural size.)

PLATE XXXI



Ruptured Pulmonary Aneurism in a Chronic Cavity.

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tuberculous disease, affording some reason for the belief that the arterial wall is first affected, leading to the production of an aneurism which nestles in a pulmonary recess or cavity of its own formation. In a case of this kind recorded by the late Dr. Cayley,[6] which one of us had the opportunity of seeing, profuse and recurrent hæmoptysis was for some few weeks the only evidence of pulmonary disease. Death occurred from early and acute disseminated tubercle.

Aneurism of the pulmonary artery may present through the wall of a bronchus, as in a case recorded by one of us in the *Pathological Society's Transactions*,[7] but we are not aware of any cases of aneurism of the bronchial arteries having been reported.

The majority of cases of fatal hæmoptysis have been found to be due to the rupture of a pulmonary aneurism in a cavity. Ulcerative erosion or rupture of large pulmonary vessels within cavities accounts for the remainder.

Bronchial hæmorrhage to any serious degree is now generally regarded as of rare occurrence. Slight hæmoptysis of this type may be observed in cases of bronchitis, and in the plastic form, accompanied by violent paroxysms of coughing, the loss of blood may be considerable. It may occur also in association with the hæmorrhagic diathesis, in purpura, and in certain malignant fevers, especially variola. Sometimes it is met with in gouty subjects and in alcoholism, associated with a heightened general blood-pressure.

Syphilitic or tuberculous ulceration of the air-tubes leads frequently to slight hæmoptysis, more rarely to copious hæmorrhage from erosion of a large bronchial or pulmonary vessel. A case once came under our notice in consultation in which very profuse hæmoptysis, which had been regarded as due to the rupture of an aortic aneurism, was present. Only slight physical examination was possible at the time, but no sign of aneurism could be found; the presence of a greatly enlarged liver suggested the possibility of syphilis, and the diagnosis was hazarded of a syphilitic ulceration of a main bronchus causing the hæmorrhage. Vigorous mercurial inunction and the administration of iodide of potassium internally resulted in the recovery of the patient, and the syphilitic view was afterwards fully confirmed.

Although it is not uncommon for hæmoptysis to be appar-

ently called forth by some unwonted effort or excitement, it is by no means so caused in the majority of cases. Dr. Wilks drew attention to the frequency with which hæmoptysis occurs during the night, and perhaps two-thirds of the cases may be said to occur during quietude, and the rest, with but rare exceptions, only during that degree of physical effort or mental excitement usual in ordinary life.

Symptoms.—Hæmoptysis is too obvious a symptom to require description. The blood expectorated may amount to no more than the slightest streaking or staining of the sputum, or several pints may be brought up.

In cases of decided hæmoptysis the patient is conscious of a gurgling sensation in the bronchial tubes, upon which, with a succession of short coughs, the blood is expelled in red, more or less frothy sputa. When more profuse, the blood may pour from the mouth in a stream, only partially interrupted by short gasping coughs. Such hæmorrhage may prove at once fatal from its very profuseness, but it is more common in fatal hæmoptysis to observe only a comparatively small amount of blood actually expectorated, the patient at once succumbing to faintness and suffocation from the overwhelming of the air-passages with blood. These latter phenomena of sudden and fatal hæmorrhage scarcely ever occur except in tuberculosis of some standing, which has resulted in pulmonary excavation and erosion or aneurism within a cavity.

The blood in hæmoptysis is usually bright red, frothy, coagulating in the receiving vessel in flattened lumps. When very copious, it may at the moment of expulsion be dark and venous-looking, but this is exceptional. After the occurrence of an hæmoptysis, the sputa are blood-stained for some hours, or two or three days, the colour being at first bright and then becoming darker. Sometimes dark, grumous-looking clots are expelled in small quantity from the lung without any previous hæmoptysis, a slight oozing having taken place, and the blood being retained for some time before expectoration.

In cases of hæmoptysis the shock to the system is often marked, especially in first attacks. The patient is agitated and alarmed, the features pallid, the expression anxious, pulse feeble and small, voice partially suppressed, temperature lowered even to subnormal. Reaction soon takes place, however, and, especially under the often injudiciously restorative

PLATE XXXII

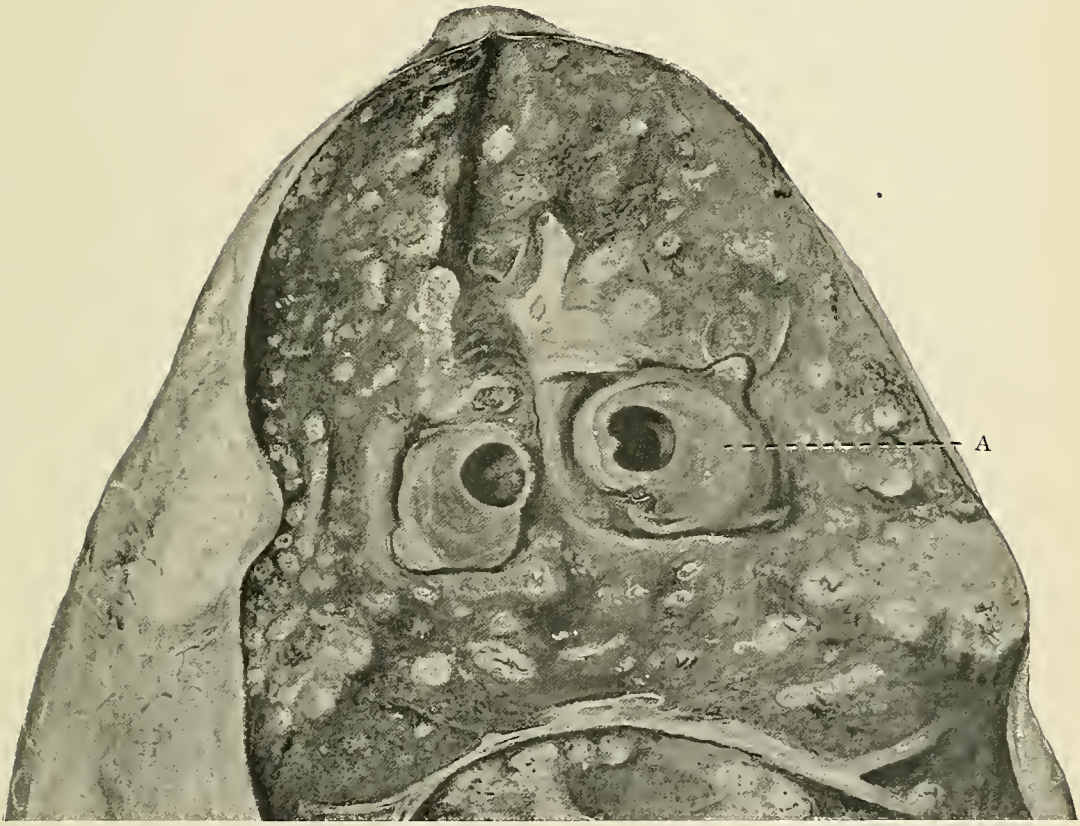
PULMONARY ANEURISM ASSOCIATED WITH RECENT TUBERCULOUS DISEASE

The drawing shows the upper portion of the right lung. This is the seat of much scattered tubercle of recent date and in the stage of caseation. In the centre of the lobe is a cavity the size of a small marble, completely filled by an aneurism (A), which is unruptured, and had given rise to no signs during life. The two halves of the aneurism are shown in section.

From a man aged twenty, a shop-assistant, who suffered from chronic pulmonary tuberculosis of two years' duration, and who died from the rupture of a second aneurism, situated in a chronic cavity in the left lung, which is figured in Plate XXXI.

(From the Brompton Hospital Museum. Natural size.)

PLATE XXXII



PULMONARY ANEURISM ASSOCIATED WITH RECENT TUBERCULOUS DISEASE.

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treatment of anxious friends, is ominous of fresh hæmorrhage. The pulse becomes large, jerking, and more or less dicrotic, the face flushed, and the conjunctivæ glistening. The sense of weakness and prostration after early hæmoptysis is often prolonged, and bears no necessary relationship to the amount of blood lost. The mental effect is also sometimes great; we have known even functional paraplegia to ensue after a slight hæmoptysis.

The temperature, as already remarked, is almost invariably depressed at the first outbreak of hæmoptysis. Recovery, however, soon ensues, and the temperature returns to its former level—febrile in those with active disease, normal in cases of quiescent phthisis. In apyrexial cases the temperature may be sometimes observed to rise about the third day, and this may be then attributed to secondary broncho-pneumonia from inhalation of blood and septic matter.

Diagnosis.—The diagnosis of hæmoptysis by a skilled observer present at the time of its occurrence can never be difficult.

1. The blood is distinctly coughed up, is more or less aërated, and either pure or mixed with expectoration.

2. After hæmoptysis there is generally a staining of the sputa for a few hours or days.

3. Blood expectorated from the lungs is never watery and non-aërated.

4. In doubtful cases inquiries should be made for epistaxis, and the gums and pharynx carefully examined (see Spurious Hæmoptysis).

5. Hæmatemesis is frequently associated with a straining, retching cough; but the history of the case, the colour of the blood, and the absence of chest signs, are sufficient for diagnosis. In hæmoptysis the blood is usually alkaline; in hæmatemesis it is always acid.

6. Only the gentlest possible measures in the way of physical examination are justifiable in cases of hæmoptysis. Percussion must be altogether avoided. The heart-sounds should be listened to, and the breath-sounds over the front of the chest auscultated, without, however, allowing any deep inspirations or other efforts on the part of the patient; by means of a flexible stethoscope the bases of the lungs may be sufficiently explored without disturbing his position.

There will usually be no difficulty in recognising at one or other apex more or less crepitant or bronchial gurgling râles, significant of the source of bleeding.

The pulse and temperature should be carefully noted, as they furnish the needful indications for treatment.

Prognosis.—In all cases of early or primary hæmoptysis a hopeful immediate prognosis may be given, since it is the rarest possible occurrence for such attacks to prove fatal. In hæmoptysis occurring in a case of quiescent phthisis in which there are known to be excavations present, the immediate prognosis must be guarded; but however profuse the hæmorrhage, an absolutely fatal prognosis is never justifiable. The fact of the first outburst not having proved immediately fatal always suggests the possibility of recovery, for it is certain that aneurismal hæmoptysis is sometimes completely recovered from, the aneurism eventually becoming firmly consolidated.

An ultimate prognosis is a much more responsible matter, and should never be given until the patient has sufficiently rallied to allow of a careful physical exploration, and a survey of all the facts of his case.

Excluding heart disease, mechanical injury, overstrain as from whooping-cough, acute sthenic pneumonia, and morbid blood conditions, hæmoptysis in the vast majority of instances means the presence of pulmonary tuberculosis, and it is one of its most important positive signs. Numbers of people doubtless make a complete recovery after hæmoptysis, but such recovery is only permanent in those of good constitutional resistance, who can and will accept the warning and place themselves under new and more suitable conditions of life. Lightly considered and carelessly treated, hæmoptysis is but the precursor of grave destructive disease.

Recurrent Hæmoptysis.—The phenomena characteristic of recurrent hæmoptysis are:

1. The presence of a localised pulmonary lesion presenting no symptoms of activity. 2. Repeated attacks of sudden and severe outbursts of hæmorrhage at short intervals, not preceded by any febrile symptoms, and not necessarily attended or followed by any extension of disease.

We may briefly allude to the following case as illustrative of this form of hæmoptysis:

In May, 1867, there first came under the notice of Dr. Douglas Powell at the Brompton Hospital a man, aged twenty-seven, described as a fitter, who had been ailing for some years with occasional cough. He complained of pain in the chest and bad cough, but, he stated, without expectoration; he had had streaky hæmoptysis several times. He was doubtful whether he had lost weight; the appetite and digestion were good, the bowels regular, and the pulse slow. His father, an intemperate man, had died of consumption at the age of forty-four. The patient himself had always been a tolerably steady man of very active habits. He was of sanguine temperament, clear complexion, medium height, and slight though robust build. A striking feature about him, and worthy of note, was his extreme excitability—an almost superfluous energy of character, which led him to do everything with exaggerated effort.

At the date of his first attendance there was present at the left apex some dulness, with a few clicks.

On June 28 he had considerable hæmoptysis, which was repeated on August 3, and again on the 8th, when a pint of blood was brought up, and a note was entered of the existence of a small vomica at the left apex. From this date the hæmorrhage continued in smaller quantities until the 17th, when it gradually subsided. It was on this date that, having regard to the continuance of the hæmoptysis and its repeated occurrence at intervals, together with the absence of any corresponding progress in the pulmonary physical signs, which were still limited to the summit of the left lung, the existence of a small aneurism of a pulmonary vessel in this situation was first suspected. He ceased attendance at the hospital at Christmas of the same year, having greatly improved in health.

The patient continued "well," as he expressed it, and at work, until October, 1868, when he returned to the hospital with slight cough, and stating that he had recently expectorated blood, but not so much as on previous occasions.

At this date there was "dulness anteriorly on the left side to the mamma, with high-pitched bronchial breath-sound, pectoriloquy and cavernous cough; sounds dry; some crepitus at angle of left scapula."

He had another slight attack of hæmoptysis in November, and at Christmas was admitted into the hospital, where he remained a month, during which time he was frequently cautioned against displaying so much energy in doing the most trivial thing, and coughing with such unnecessary violence. He had no appreciable expectoration, and left the hospital feeling well.

No pyrexial symptoms had ever been observed in this patient, and during a slight attack of hæmoptysis, whilst in hospital, the temperature was observed to be normal.

He was not again seen until August, 1870, when, having remained quite well and at work until a few days previously, he expectorated half an ounce of blood. The physical signs were still limited to the apex.

The patient ceased attendance in October, and continued pretty well until March 4, 1871, when he again attended with hæmoptysis. Although still spitting blood freely, he, quite against orders, attended personally from Battersea on March 8, and brought up a considerable quantity of blood in the out-patient room. This attack proved the most prolonged and desperate one he had yet had, and nearly terminated fatally. The hæmorrhage continued with frequent outbursts of half a pint until the 30th, from which date the attacks abated in violence, apparently rather from lack of blood-supply than from the efficacy of remedies, which, however, were steadily persisted in until April 13, when there had been no considerable hæmoptysis for a week.

He again attended personally, though with great difficulty from his extreme weakness, on May 4. At this date there was noted at the left apex "retraction of lung, dulness, cavernous respiration, and rhonchus (slight)." Posteriorly there was "diffused crepitation, with some defective resonance." This was the first occasion on which the lung had appeared to suffer from the effects of hæmoptysis.

The cough was troublesome, especially in the morning; and on the 11th he was ordered ether and ammonia expectorant, lest his violent and unaided efforts at expectorating should lead to a fresh opening of the probable aneurism, which seemed to be the only conceivable source of such profuse and repeated hæmorrhage. It was extraordinary to note the rapidity with which the patient regained flesh, strength and colour, although butcher's meat was only allowed every other day; stimulants were cut off, and abundance of milk alone permitted. He continued to take mineral acids and cod-liver oil. He did not at all approve of the diet; but from previous experience of his rapid blood-making qualities, one was convinced that a more generous regimen would have led to a return of the hæmorrhage.

On June 8 he had one comparatively slight attack of hæmoptysis, and on the 29th the physical signs showed enlargement of the right lung, the margin of which reached across the median line; there was still some irritative bronchitis at the left base, indicated by diffused submucous râles.

December, 1871.—Beyond an occasional tinge in the morning expectoration, the patient had had no more hæmoptysis, and had returned almost to his usual health, though the breath was shorter. Since June he had taken no oil, but for a few weeks some digitalis was added to his mixture. The restricted diet had been continued, though less so of late.

This man did not attend the hospital again, but was seen about occasionally, and apparently well, during the next five years. The case exemplifies well the main features of recurrent hæmoptysis—viz.: (1) repeated copious hæmorrhage, obviously arising from disease localised at one portion of the lung; (2) pulmonary disease, chronic in its course, and but

little influenced directly by the hæmorrhage; (3) the hæmoptysis, *though it may prove directly fatal*, being accompanied by no severe fever or secondary pneumonia, and from it the patient frequently making a speedy recovery.

The pathological condition common to all these cases of recurrent hæmoptysis is that of a slowly forming cavity, or one formed by a very localised process of an active character, in the walls of which pulmonary vessels still patent are exposed. On such a vessel an aneurism then forms, projecting on the cavity side of the vessel. It will be observed that the case above described did not begin with hæmoptysis; the man had had some dry cough, and occasional streaky sputum, for some years previously; and a few days after the first considerable hæmorrhage a vomica was found at the left apex, where some two months previously there was consolidation and softening.

All cases of this kind are, however, not necessarily tuberculous. For example, we have met with an aneurism in a bronchial dilatation, and in another instance an abscess in the lung was attended with all the features of pulmonary aneurismal hæmoptysis, from which the patient ultimately recovered.

The danger in cases such as these is from the rapid outburst of blood, which at once floods the bronchi and may asphyxiate the patient. In other cases the actual amount of hæmorrhage is the cause of death on the first occasion. Nothing is, however, more striking than the recovery of some patients from what appears to be the most hopelessly profuse hæmoptysis, Nature apparently seizing the moment when, from faintness, the blood is at a standstill, to heal the breach by the formation of a coagulum. Hence the importance in treating such cases of withholding all stimulants till the latest moment. Rokitansky refers to another mode of arrest of the hæmorrhage from a large vessel in a cavity—viz., by the cavity becoming blocked by coagulum, which thus compresses the vessel. We have seen an instance, post-mortem, in which the apex of the right lung was converted into a blood-cyst as large as a lemon, which was quite closed, and which had been produced by hæmorrhage into a cavity.

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[3] "Varicose Aneurysmal Dilatation of Two Small Branches of the Pulmonary Artery. Rupture of One of Them. Death by Sudden Hæmoptysis in a Case of Phthisis," by Dr. Quain, *Transactions of the Pathological Society of London,* 1866, vol. xvii., p. 79.

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CHAPTER XXXIX

ON FALSE OR SPURIOUS HÆMOPTYSIS

By true hæmoptysis is meant, as we have seen, hæmorrhage from or through the lungs; either from the lung texture proper, or from the lining of the bronchial tubes ramifying through the lungs, or more rarely from some external source, the blood being discharged through the lungs.

By false or spurious hæmoptysis is meant the spitting of blood which has escaped from some portion of the mucous membrane lining the nasal, buccal, or pharyngeal passages. Perhaps the true anatomical line of division between true and false hæmoptysis would be at the glottis, for below this point the mucous membrane assumes the ciliated columnar epithelium characteristic of the bronchial tract, whilst above the epithelium is of the squamous kind. In true hæmoptysis, with the exception of those rare cases in which the hæmorrhage comes from the larynx or trachea, or from some external source the blood escapes from the pulmonary or bronchial vessels; in false hæmoptysis from branches of the carotid trunks.

The parts whence the blood of false hæmoptysis is usually derived are the nasal mucous membrane, the pharynx, the gums and dental alveoli. The following are the more important conditions under which this phenomenon is met with.

1. In cases of decided epistaxis some of the blood commonly trickles down the back of the throat, and excites cough, by which it is removed in clots and with staining of the saliva; the source of hæmorrhage is obviously, however, the nasal membrane, and no real difficulty in diagnosis ever arises. It is only in cases in which the nasal hæmorrhage is but slight, and attended with little or no escape of blood through the anterior nares, that there is any probability of the affection being mistaken for hæmoptysis. This occurrence may happen

at night, and the patient wake up spitting blood. The absence of fever and of pulmonary physical signs, and the detection of blood mingled with the nasal mucus when expelled, or of coagula in the nasal passages, will render the diagnosis in these cases also clear.

2. Ulceration of the throat, especially when malignant, may lead to copious hæmorrhage, and in these cases, again, no difficulty is likely to arise in the way of diagnosis.

3. A class of cases is now and again met with which occasions much trouble to the practitioner, and requires decision in management. These are cases of feigned or hysterical hæmoptysis. Such are nothing more nor less than downright attempts at imposition, the blood being produced either by sucking the gums, or by pricking or incising them. The late Sir George Johnson[1] referred to the case of a young girl sent up to King's College Hospital by a lady interested in her, with an elaborate history of symptoms, including blood-spitting. The character of the expectoration, which consisted of unaërated saliva mixed with fresh blood, was sufficient to indicate its source, and on examining the mouth with a bright light, about twenty fine cuts or scratches were discovered on the mucous membrane covering the hard palate. A sharp reprimand and a short course of shower-baths and steel tonics speedily removed the symptoms.

In some hysterical cases, however, we have known the blood-stained saliva to be highly aërated, probably produced by sucking the gums after injuring them with a needle. The appearance and the physiognomy of the patient are generally sufficient to excite suspicion, and other hysterical symptoms are usually present.

4. A morbid state of the gums, a degree of pyorrhœa alveolaris, frequently arises from want of due attention to the teeth, or from the presence of decayed stumps in the alveoli. On gently pressing the edge of the gums, pus escapes from beside the affected teeth, and on the slightest touch or friction blood exudes from the mucous membrane, which is swollen and congested, a livid line running along the margin of the gum. A similar condition arises from the effects of certain drugs, especially mercury and, to a much less degree, lead and iodide of potassium. In these cases there is fœtor of breath, and an inspection of the gums and teeth at once suggests the

probable source of the blood-spitting, which is usually insignificant in amount, and unaccompanied by any cough or chest symptoms.

The treatment of these cases falls partly within the province of the dental surgeon; it consists in the employment of astringent tooth-powders, of which one of the best for the purpose is composed of finely powdered kino one part, to three or five of prepared chalk, with or without a little animal charcoal. Another preparation which we have found of much value in cases of pyorrhœa is a lotion composed of liquefied carbolic acid, three minims; liquor potassæ, three minims; and rosewater to the ounce. This should be applied to the gums on a pledget of cotton-wool several times a day. Any decayed teeth must be removed or stopped. More or less dyspepsia is usually present in these cases, partly arising, no doubt, from the condition of the gums and teeth, and a stomach cough, added to the staining of the sputum, may suggest to the patient that he is consumptive.

5. An insufficient supply of vegetable food, a common dietetic error among all classes of people in towns, leads to a spongy, congested state of the mucous membrane of the mouth and fauces, of the same kind as that which, in a more intense degree, is associated with the other lesions characteristic of scurvy. This is one of the common causes of spurious hæmoptysis. The relaxed condition of the throat, resulting in the secretion of an undue amount of viscid mucus, gives rise to cough, and the mucus expectorated, or rather hawked up, from the pharynx, together with the saliva from the mouth, is from time to time tinged with blood. It is often difficult to distinguish this form of false from true hæmoptysis. Indeed, the condition of the mouth is but a sample of that of the mucous membranes generally; and the larger bronchi, if affected with catarrh, are apt to yield a viscid and slightly stained secretion. We have observed in some cases of phthisis a staining of the expectoration, which has seemed to have arisen in the same way.

In the cases under consideration there are no pulmonary signs to be discovered even after a very careful examination. The hæmorrhage is never in large quantity, and it consists of a tingeing or streaking of sputum which is distinctly made up of mucus mixed with saliva, giving rise to a dirty red fluid

containing some little streaks or clots of blood. On microscopic examination, squamous epithelium cells are seen in abundance, and red blood-corpuscles are but thinly scattered over the field. The patient complains of the taste of blood in the mouth, and this is especially disagreeable after sleep. The nutrition is not good, the muscles are flabby and wanting in tone, and the patient feels languid and out of sorts. There is commonly some anæmia present.

A most favourable prognosis may confidently be given in these cases, if we are quite satisfied as to the absence of any pulmonary sign.

In treatment the diet must be attended to, an abundance of fresh fruit and vegetables being added. Five or ten grains of citrate of iron should be ordered in fresh lemon-juice two or three times a day, and cod-liver oil may often be given with great advantage. Some tannin solution should be used as a gargle, and to rinse the mouth. A pleasant mouth-wash and gargle to use night and morning consists of Hazeline ℥ii., Glycerini Boracis ℥ii., Eau-de-Cologne ℥i., Aquam Rosæ ad ℥viii.; one tablespoonful to a wineglass of water to rinse the mouth, and some undiluted lotion dropped on to a soft badger's-hair toothbrush previously dipped in water, to brush the gums.

In cases of phthisis in which we suspect this morbid condition of the bronchial mucous membrane to be present, fresh lemon-juice, a not unpleasant vehicle for cod-liver oil, is valuable.

6. In certain cases of anæmia, attended with all the other phenomena of that disease, the mucous membrane of the mouth and fauces, although pallid in appearance, exudes a sanguineous fluid, which, mixed with the saliva, causes spurious hæmoptysis. There is in these cases, so far as one can discover, no definite bleeding-point to be seen, but in the course of twenty-four hours a considerable amount of blood will transude through the vessels. The transudation is ordinarily very slow, and in the daytime is scarcely noticed; but during the night some accumulation takes place, and, on waking, the patient expels perhaps an ounce or more of bright red unaërated fluid, containing a few coagulated films, giving an appearance closely resembling that of currant jelly and water. Some of the sanguineous fluid often escapes from the

mouth upon the pillow during sleep. The patients suffering from this affection are mostly females. Amongst other symptoms of anæmia the menstruation is disordered or suppressed, and commonly, but not always, at the menstrual period the escape of blood from the mouth is considerably increased. Probably from the same cause—an increased blood-pressure finding relief at the surface of least resistance—any extra exertion is apt to produce an increase in the sanguineous flow. The real pathology of these cases is, however, confessedly obscure; but this is the variety of false hæmoptysis for which we are most often consulted. The patients are short of breath; they sometimes have a hard cough, and complain of pain in the left side and considerable prostration, which symptoms, with increasing pallor and blood-stained expectoration, are quite sufficient to persuade them and their friends that they are consumptive.

In such cases the greatest pains must be taken to exclude the presence of tuberculosis. The sputum, however scanty, must on several occasions be carefully examined for tubercle bacilli, if necessary, using concentration methods (p. 574), and a watch kept upon the patient's weight and temperature. As regards physical diagnosis, the respiratory sounds will usually be found to be weak and partly suppressed from want of muscular power. The percussion is, however, even on the two sides, and the respiration, although feeble, is vesicular. The character of the cough, both as heard through the stethoscope and otherwise, is usually quite distinct from that of chest disease. Moreover, all the signs of anæmia are present, venous hum, arterial murmurs, and so forth, and the "pain in the chest" is, without much difficulty, ascertained to be either inframammary neuralgia or gastrodynia. Some patients with this form of spurious hæmoptysis have plenty of colour in the cheeks, and are plump rather than emaciated; but they nevertheless present other evidences of anæmia. The careful observation of a large number of these cases for long periods enables us to say that it is very unusual for them to develope phthisis.

The condition calling for *treatment* is the anæmia. More fresh meat must be taken, and, if necessary, some hydrochloric acid and pepsin added to aid digestion. In some cases there is considerable disorder of stomach present, which must be

first set right before the remedies appropriate to anæmia can be given. These remedies are the astringent forms of iron. Cold salt baths or sea-bathing allowed only for a very brief time—one or two minutes—and immediately followed by vigorous friction, are most useful in the convalescent stage. Abundance of fresh air and out-of-door exercise is, of course, to be insisted upon. The patients and their friends are often much afraid of fresh air, and the cases have usually at the period at which they come under observation been aggravated by confinement in warm and ill-ventilated rooms. If, as often happens, there are decayed teeth present, setting up irritation in the gums, and increasing the disposition to hæmorrhage, these should be attended to. Some calcium lactate may be given with syrupus calcii lacto-phosphatis, and a little reduced iron with the meals.

7. General hæmorrhage from the whole mucous membrane of the mouth is sometimes seen in hæmophilia. As in the variety described under the preceding heading, no perceptible lesion is discoverable in the mucous membrane, but the hæmorrhage in this case is usually so considerable in amount as to prevent any possibility of mistaking its nature, and other signs of the disease may be present.

Such are the more important conditions under which spurious hæmoptysis is met with. In all cases, however, in which the cause is not apparent, a very careful examination of the pharynx, back of the tongue, and of the laryngeal region should be made with the laryngoscope. In this way it has happened that a small area of ulceration has been found, for example, on the posterior aspect of the tongue, on treatment of which the blood-spitting has ceased.

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CHAPTER XL

ON OTHER IMPORTANT COMPLICATIONS OF PULMONARY TUBERCULOSIS

Tuberculous Meningitis.—This fatal complication of pulmonary tuberculosis occurs as part of a secondary and generalised miliary infection. When the meninges of the brain are involved in this outbreak of tubercle, the special symptoms that arise are so grave as to set aside from view all other conditions present.

A notable feature about tuberculous meningitis, when it complicates phthisis, is the uncertainty and insidiousness of its supervention. It is happily a somewhat rare complication —occurring in only 7 of our 275 autopsies[1a]—yet there is no case of phthisis, and no stage of any case in which the conditions for its possible occurrence are not present Thus a child may have a small apex lesion, which has resulted in some local induration and flattening, with complete subsidence of all symptoms; she has steadily improved, and is regarded by her parents as well, although the doctor pronounces her chest still to be delicate, when suddenly she developes brain symptoms, which terminate fatally within three weeks. Such is a common history.

We will briefly relate one or two cases which may be regarded as typical of their kind.

Case 1.—George B., aged twelve, a thin, pale, neglected-looking boy, was admitted into hospital on August 26 with a history of recent bronchitis and presenting the remains of a broncho-pneumonia, with some hectic symptoms. On September 18 vomiting commenced, occurring after food, and attended with some diarrhœa, a coated tongue, and a slight rise of temperature (99°). The vomiting became more frequent, and persisted through the next seven or eight days. The face was flushed, the skin hot and perspiring. No headache was complained of. On the third day of these symptoms the urine yielded

on boiling a heavy cloud of phosphates, a sign which we have observed in other cases of the disease. On the tenth day (September 27) the patient became unconscious, and the following note was made: "Pulse 76, *irregular*, lips dry, tongue furred. Expression of face *drowsy, suspicious*. Will not answer questions. Tries to put out tongue when sharply told to do so, but fails. Is slightly delirious. Respirations 20 in the minute, temperature 100·8°. There is slight occasional *twitching of the left arm* and pectoral muscle. When aroused drinks oatmeal-water with avidity. Evidently tries to answer questions put to him but fails to do so. At 9 p.m. twitching of both arms and legs observed, with picking at bedclothes, and grasping at nothing. Has not vomited after taking cream and brandy; pulse 80; temperature 100°."

On the eleventh day, September 28, temperature 100·2°, pulse 80, the patient more restless. Fingers continually working, scratching, or pulling at teeth. *Muscles of neck and back rigid.* At 4 p.m.: movements of arms and legs more violent. Rigidity of back more marked; temperature 102·2°; bowels open from medicine; motions loose. September 29: temperature 101·2°, pulse 80, weaker. Slight external strabismus of left eye. September 30: 10 a.m., temperature 102·4; 7 p.m., 103°, pulse 120. Eyes roll slowly from side to side, pupils dilated. October 1 and 2: morning temperature 101·6°. Emaciation increasing. Some resistance to extension of arms. Conjunctivæ more sensitive, pulse 130, *regular*. Patient lingered for a week longer in much the same state, and then sank on the twenty-first day. The post-mortem revealed the usual evidence of meningeal tuberculosis.

In this case the insidiousness of onset was well illustrated. During the first days of the attack no headache was complained of, nor did the boy at any time suffer much from this symptom. Obstinate vomiting resisting all treatment was up to the time of unconsciousness the only definite symptom, and its significance was at first obscured by other evidences in tongue and bowels pointing to gastric derangement. Later, twitchings and irregular movement of the limbs, with increasing unconsciousness, rigidity of neck, strabismus, and blindness were present in this, as in most other cases of meningitis.

Case 2.—In this case, that of an adult, aged thirty-three, with advanced phthisis, the first symptoms were headache and confusion of vision, which latter was ascribed to $\frac{1}{100}$ grain hyoscyamine given in a cough linctus. The pupils, however, were not dilated. The patient vomited twice in the course of the next two days, and on the fifth day twitchings of the flexor tendons were observed. Two days later the pupils became unevenly contracted. The patient became drowsy; he could not find words to answer questions. He died on the nineteenth

day after the commencement of symptoms, irregularity of pulse and breathing, drowsiness, and variable twitching of the limbs, being observed towards the end.

The following propositions embrace what is reliable in the diagnosis of this complication, sometimes so startling, at others so insidious, in its onset.

1. *Persistent headache and vomiting are the most common first symptoms of the disease. They may or may not be combined. They are usually associated with furred tongue and disordered bowels, which tend to mask their significance.*

The headache of tuberculous meningitis does not affect with constancy any particular portion of the head. It is sometimes frontal, often over the crown of the head, occasionally at the back or on one side. Although always a sign to cause anxiety when it occurs at all severely or persistently in phthisis, yet it is never, even when associated with vomiting, sufficient to enable us to form a diagnosis. In several cases we have found headache so severe, persistent, and, taken together with the general aspect of the patient, so apparently characteristic of meningitis, as to have led us to feel very apprehensive as to its real significance; yet again and again our suspicions have proved to be unfounded. On the other hand, in the majority of cases of true tuberculous meningitis that have come under our observation, other suspicious symptoms have been associated with the headache until the appearance of more decided signs removed all doubt. Hence *headache or vomiting, although not sufficient for diagnosis, are signs which, if not readily relieved by treatment, should always arouse grave suspicions.*

2. *Disordered vision, impaired memory, and confusion of ideas are signs which, taken in association with headache, are almost diagnostic; the supervention of muscular twitchings or convulsions is of the highest significance.*

Any or all of these signs may closely follow the appearance of headache or vomiting. They may, one or more of them, constitute the first symptoms of the disease. Paralysis of the third or sixth nerve usually occurs among the later symptoms, when the effused lymph and contractile tissue drags and exercises pressure upon the nerves. Ophthalmoscopic examination of the fundus may yield a valuable positive result, but the absence of tubercles in the choroid signifies little, as they are

present in only a minority of cases. Thus, choroidal tubercles were found in only fourteen out of forty-seven consecutive cases (29·8 per cent.) examined post-mortem at St. Bartholomew's Hospital, and during life they are discovered even less frequently.[2]

3. *Drowsiness deepening into coma, but often with intervals of consciousness, is the most constant of the later symptoms of tuberculous meningitis. It depends upon effusion into the ventricles.*

4. *Irregular pulse and irregular respiration are amongst the occasional early signs of tuberculous meningitis.*

Both irregularity of the pulse and of the respiration are not uncommon after the period of coma, but they are then signs of little importance. A marked irregularity of the pulse occurring, however, early in the attack is of greater significance than is usually recognised. The pulse is commonly rather slow than quick, sometimes markedly infrequent. The respirations are rarely affected during the early stages of the disease.

5. *The temperature is as a rule not much elevated. It is more often raised towards the end of the attack, its rise being apparently associated with secondary inflammatory lesions. It is of little or no value in diagnosis.*

6. *Kernig's Sign.*—A common symptom met with in meningitis is that known as Kernig's sign. By this is meant the inability to straighten the patient's leg when the thigh is placed at a right angle to the trunk, owing to contraction induced in the calf muscles. The sign is not, however, limited to meningitis, and its diagnostic value is correspondingly diminished.

7. *Lumbar Puncture.*—In cases of doubt the diagnosis is much aided by a lumbar puncture. If tuberculous meningitis be present, the fluid withdrawn will be found to be clear or faintly turbid. The albumin-content is increased, so that no longer a "faint haze" is observed, but a definite ring at the line of junction when the cold nitric acid test is applied, and on boiling with an equal quantity of Fehling's solution no reduction is now obtained. If a film from the deposit obtained by centrifugalisation be stained, *lymphocytes* will be usually found to form the great majority of the cells present. When cultivated on ordinary media the fluid is sterile, but

tubercle bacilli may often be demonstrated if the small fibrinous clot, which in many cases forms on standing, be teased out on a slide and stained by the Ziehl-Neelsen method, as recommended by Dr. Graham Forbes.[3]

In these respects tuberculous meningitis differs from other varieties of acute meningitis. In the latter the fluid will be found more turbid, owing to the greater number of cells present, and the differential count shows that *polymorphonuclear leucocytes* predominate. On staining films various organisms, whether meningococci, pneumococci, streptococci or others, will be observed, and their presence will, as a rule, be confirmed by cultivation. In health the cerebro-spinal fluid is clear, and almost free from cells.

8. The blood-count shows, as a rule, a *leucocytosis*, the increase in number of the white cells, which may early in the course of the disease number 12,000, being chiefly due to the presence of polymorphonuclear cells.

The *duration* of this disease is varied; it may terminate in a few hours, or days, or weeks, and is generally fatal before the twenty-first day. Patients will sometimes linger for many days in a state of complete insensibility from effusion into the ventricles. It is not infrequent for a deceptive amendment of symptoms of short duration to take place before the fatal issue.

Lardaceous Disease.—In about 10 per cent. of cases of phthisis lardaceous changes are found at the autopsy in certain organs. The table on p. 568 shows, in detail, the results obtained in 263 cases of chronic pulmonary tuberculosis upon which autopsies were performed by one of us at the Brompton Hospital during the years 1900 to 1903.[16] In these statistics, it should be added, we are alluding to the coarse degrees of lardaceous degeneration demonstrated to the naked eye by the iodine test; in a considerably larger percentage microscopic examination would have revealed a trace of the condition.

The figures given below closely agree with those quoted by Sir J. Kingston Fowler[4] from records taken at the same hospital during the years 1893-94. It would seem, however, that formerly lardaceous disease was more often met with. Thus, in ninety-nine post-mortem examinations, made consecutively at the Brompton Hospital by one of us in the years 1869 to

1875, it was found in twenty cases, eight male and twelve female, or in about 20 per cent. of the whole, a difference in percentage from that in the table below, which is possibly to be attributed to the more hygienic conditions under which the disease is now treated, and the diminished liability, therefore, of the patients to be infected with virulent secondary organisms. In the twenty patients just referred to the pulmonary disease had a maximum duration of sixty-six months, a minimum of four months, with a mean of twenty-six months. The spleen was affected in nineteen of the twenty cases.

TABLE SHOWING THE FREQUENCY OF OCCURRENCE OF LARDACEOUS DISEASE IN CHRONIC PULMONARY TUBERCULOSIS, AND THE ORGANS CHIEFLY AFFECTED, BASED UPON 263 CONSECUTIVE AUTOPSIES (188 MALES, 75 FEMALES).

	Males.	Males per Cent.	Females.	Females per Cent.	Total.	Total per Cent.
Present in	18	9·5	10	13·3	28	10·6
Analysis of above—						
Spleen affected ...	14	7·4	8	10·6	22	8·3
Kidneys ,, ...	11	5·8	7	9·3	18	6·8
Liver ,, ...	11	5·8	6	8·0	17	6·4
Intestines ,, ...	7	3·7	6	8·0	13	4·9
Stomach ,, ...	4	2·1	3	4·0	7	2·6

From the figures above quoted it will be seen that in lardaceous disease following tuberculosis of the lungs the spleen is the organ most commonly affected, and after this the kidney and liver in about equal proportions. Less frequently the intestines, stomach, and suprarenals, and occasionally other organs—such as the lymphatic glands, the thyroid, testes, etc. —show the characteristic change. This especial vulnerability of the spleen is, perhaps, peculiar to cases originating in phthisis; for we may remark that in 118 cases collected at St. George's Hospital by the late Dr. Dickinson,[5], of which forty-three at most originated in phthisis (suppuration of various kinds and syphilis accounting for the remainder), the order of frequency was as follows: kidneys affected in 95 cases; spleen in 76; liver in 65; intestines in 35; stomach in 9.

The lardaceous change in phthisis is no doubt connected with the suppuration which is taking place in the lungs and the imperfect drainage and consequent absorption of toxines, which is inseparable from this condition. In this connection

it is interesting to recall that the change has been produced experimentally in animals by Krawkow, Alan Green,[6] and others, as the result of the inoculation of attenuated cultures of pyogenic organisms or their toxines.

Lardaceous disease may be recognised as a complication in phthisis by the detection of an enlarged spleen, which presents below the cartilages, firm and smooth on palpation. The liver also may be found large, smooth, and hard, with well-defined and rigid marginal outline. In many cases albuminuria is the first sign, considerable in amount, and accompanied by hyaline casts and a variable degree of dropsy.

It only rarely happens, and chiefly in the fibroid variety of phthisis, that the lardaceous disease causes death, and in these cases the fatal result is due to kidney complication. In a few instances the intestinal mucous membrane is gravely involved, and obstinate diarrhœa exhausts the patient. The appearance of the change is often associated with chronicity.

Albuminuria.—The presence of albuminuria is rare in the early stages of phthisis. Later on it may result from simple chronic nephritis or lardaceous degeneration, both, however, less common now than formerly, or tuberculous disease of the kidney and pelvis (or other portion of the urinary tract), the so-called scrofulous disease of the kidneys described by earlier writers.

(*a*) *Lardaceous degeneration* of the kidneys is not common in phthisis, and is somewhat less frequently observed than a similar affection of the spleen. It occurs in the more chronic cases, and is often associated with a like affection of other organs.

A considerable degree of renal dropsy may attend the albuminuria arising from this cause, and the abdomen is generally enlarged from the presence of an amyloid or amylo-fatty liver. Diarrhœa is often a marked symptom in this form of phthisical albuminuria, and sometimes uræmic vomiting and convulsions occur, but much less frequently than in other forms of nephritis.

(*b*) Another cause of albuminuria and renal phenomena in tuberculosis is a form of nephritis, parenchymatous in nature, in which the kidney is somewhat enlarged, its capsule adherent, the organ softer than natural, and less dry on section, presenting a swollen cortex of a mottled appear-

ance from points and streaks of fatty degeneration. This kidney does not give the amyloid reaction. The albuminuria is considerable in amount, and there is a tolerably copious sediment from the urine, containing abundant epithelial and fatty casts.

This form of renal complication is perhaps of less frequent occurrence than formerly. It is much more serious than the lardaceous form, being attended with scantiness or suppression of urine, obstinate and exhausting vomiting or diarrhœa, or pulmonary œdema and asthmatic phenomena, and other grave uræmic symptoms.

(*c*) The kidneys necessarily partake in the pathology of acute miliary tuberculosis, and in patients dying of chronic phthisis the occurrence of a few miliary tubercles in the kidneys is by no means uncommon. In our own autopsies we found such tubercles, visible to the naked eye, in 28 out of 263 cases of chronic pulmonary tuberculosis, a percentage of 10·6; whilst Dr. Joseph Walsh,[7a] examining 60 cases, was able to demonstrate their presence microscopically in 35 (58 per cent.). In conformity with these results, we may add that Drs. Ravenel, Walsh, and Smith[7b] have proved that in advanced phthisis tubercle bacilli not infrequently pass into the urine, and by inoculating guinea-pigs with the washed sediment of two litres, they were able to demonstrate their presence in 14 out of 17 cases. Bacilli may thus find a nidus in the kidney, and give rise to a few miliary tubercles. Ulcerative tuberculosis of the organ, however, with its attendant caseating masses and vomicæ, is rare as a complication of phthisis. Out of 263 autopsies, we only found such a complication in 6. Its occurrence is indicated by lumbar pain, increased frequency of micturition, and especially by the presence of a thick deposit of pus in the urine, in which tubercle bacilli will be found. Like some other tuberculous manifestations, it is more common as a primary local affection, upon which pulmonary disease may later be engrafted.

The occurrence in the urine of the ordinary diazo-reaction or of the dimethyl-amido-benzaldehyde reaction, introduced by the late Professor Ehrlich,[8] possesses little clinical significance.

Fistula.—*Fistula in ano* occurs in a small proportion of cases of pulmonary tubercle, and in our series of 263 autopsies

it was only observed on three occasions.[1c] In our experience the complication is almost entirely limited to males.

The same causes which give rise to fistula in the healthy subject may produce it also in the sufferer from phthisis. It may thus be sometimes traced to fish or rabbit bones, or other irritating objects in the bowel, or result from the exposure of the parts to cold and wet.

Two special methods are described by authors by which this complication may arise—viz.: (1) From tuberculous infection of the rectal mucous membrane by swallowed sputum or ulcerous discharges from a higher portion of the bowel. A local ulceration is thus set up, which, by extension outwards, causes ischio-rectal abscess. (2) In other, and probably more numerous cases, the tuberculous disease commences in the submucosa, extends outwards into the ischio-rectal fossa, and only later perforates the intestine.

The ischio-rectal abscess thus produced lacks as a rule the acute inflammatory symptoms which are generally associated with the non-tuberculous variety of the disease. It is, on the contrary, often of a chronic type, giving rise to but few symptoms, and it may not be until external rupture occurs that the patient is led to seek advice.

Treatment.—Fistula may occur early in the course of phthisis, when the pulmonary signs are but slight. More commonly it makes its appearance when the malady is more advanced and cachexia already marked. In cases of this kind the complication becomes of secondary importance, and, unless causing much additional suffering and exhaustion, it should not be interfered with by operation.

In slighter cases, each one must be judged upon its merits. If the discharge be small in amount, the patient suffering little, and the fistula causing but trifling inconvenience, we should not advocate surgical interference; for it must not be forgotten that the effect of the anæsthetic and the operation itself exercise a depressing influence upon the patient, which is not infrequently manifested by increased activity of chest disease. There is some evidence also to show that in certain cases a kind of alternation exists between the activity of the fistula and that of the pulmonary disease—an old clinical observation possibly explained by the recent researches of Sir Almroth Wright.

There are instances, however, in which the local inconvenience and suffering from the fistula and the mental distress are so great as to necessitate some operative measures, and in such cases surgical treatment must be carried out.

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CHAPTER XLI

THE DIAGNOSIS OF PULMONARY TUBERCULOSIS

In the preceding chapters we have considered the various clinical manifestations of phthisis which may be met with, and the more important complications of the disease. We have seen that in the great majority of cases the tuberculous process begins near the apices of the lungs, and extends downwards, while the physical signs, not very distinct at first, gradually become more and more definite as consolidation, softening and cavity formation succeed each other, until finally the patient presents the classical picture of advanced disease so graphically portrayed for us by Aretæus[1] many centuries ago. In such a case diagnosis is no longer difficult. But, in order that treatment may be effective, it is of the first importance that the disease should be recognised at an early stage, and how this may be effected we propose to consider in the present chapter.

Upon the methods of physical diagnosis which we have already discussed great reliance may be placed. With commencing consolidation, which is associated with effacement by collapse or thickening, of the vesicular tissue of the lung over the affected area, we obtain a slight impairment of the percussion resonance, and with it a weakening of the breath-sound and a partial or complete suppression of its vesicular quality, the expiratory murmur becoming more prolonged than natural. As the consolidation becomes more dense, the breath-sounds gradually assume a bronchial character, both inspiratory and expiratory sounds being conducted from their tracheo-glottic source. Vocal resonance and vocal vibrations are both increased, and a few moist crepitations may become audible. It is with the inspiration after a cough that these crepitations are first disclosed.

Should early apical signs such as these be found in patients who have sought advice complaining of cough, wasting, or perhaps a slight hæmoptysis, they are of diagnostic impor-

tance. A rise, it may be only slight, of evening temperature is usually already to be detected, and the patient will generally complain of some night sweating. Later on, when softening of the tuberculous foci in the lung takes place, bacilli will make their appearance in the sputum; and even in a case in which the expectoration is scanty it may still be possible to find some in the small specks brought up in the early morning on first waking, which should be examined patiently for several days in succession.

If tubercle bacilli be not found by ordinary staining, *concentration methods* must be employed. These are of various kinds, but their object is the same, namely, by the liquefaction of a considerable quantity of sputum (5 to 20 c.c.) and its subsequent treatment by centrifugalisation or otherwise, to obtain a film in which all the bacilli are collected. The antiformin method of Uhlenhuth and Xylander[2] is that most generally employed at the present time. This consists in treating in a test tube equal quantities of sputum and 15 per cent. antiformin—a solution in definite proportions of sodium hypochlorite and sodium hydrate. The mixture is shaken from time to time and soon becomes homogeneous. It is then centrifugalised and the supernatant liquid pipetted off. The deposit is washed in normal saline solution and again centrifugalised. The supernatant fluid is once more pipetted off, and films, made from the deposit, are stained by the Ziehl-Neelsen method. We have on several occasions known tubercle bacilli to be thus discovered when ordinary methods several times repeated had failed.

The antiformin method may also be applied to the stools, and when sputum cannot be obtained, as in children and those who swallow their expectoration, the stools may be examined, and it is sometimes possible to discover the bacilli in the fæces. In a little girl under our care at the Brompton Hospital, who presented signs and symptoms of advancing tuberculosis of the lungs, but without expectoration, tubercle bacilli were in this way easily demonstrated.

The presence of tubercle bacilli in the sputum makes the diagnosis sure, but we must not forget that their presence also indicates that the disease is no longer in its earliest stage, but that softening of the pulmonary focus has occurred, and that a case of "closed" has been converted into one of "open

tuberculosis." It is pertinent, therefore, to ask whether we have any other methods of diagnosis which may be made use of in the recognition of those very early cases of "closed" tuberculosis in which the physical signs may not be clear, the symptoms are slight and indefinite, and in which no tubercle bacilli have yet made their appearance. Let us consider the following:

X-Ray Examination.—In most X-ray photographs in the healthy adult the rays reveal an irregular shadow at the hilum, sometimes with certain deeper points of shading, these appearances indicating old lesions, tuberculous or other, of the bronchial glands, with areas of calcareous degeneration. From this central shadow there radiates through the lung a delicate arborisation corresponding to vessels and bronchi, among which the main trunks passing to the upper and lower lobes stand out more conspicuously. In cases of phthisis in the adult these appearances are visible, but in addition there is often seen a mottled shading which is characteristic of well-developed pulmonary tuberculosis, these shadows having, when *in focus*, a somewhat woolly outline when the disease is active and becoming more defined when it is of old standing. Such mottling is not, however, an early sign of phthisis, and is, in our experience, not seen without there being some physical signs to indicate the disease.

In the adult we have seen reason to believe (p. 471) that it is rare for infection of the lung to spread from the bronchial glands into the substance of the lung, where the changes would be beyond the reach of physical examination, and the X-ray appearances held to indicate this form of "hilum tuberculosis" are not to our mind convincing. On this point further correlation of X-ray appearances and post-mortem findings is much needed. We have ourselves rarely met with cases of phthisis which do not quite early give physical signs at the apices of the lungs.

Passing to the screen examination, we not infrequently find a restricted movement of the diaphragm on the affected side, and this was held a few years ago to be the earliest sign of pulmonary tuberculosis. It is now recognised, however, that this sign may occur under many conditions apart from phthisis, and its diagnostic value is not therefore great.

Another sign observed on the screen is a diminished

translucency at one or other apex and a failure of this portion of the lung to light up on deep inspiration to the same degree as its fellow on the unaffected side. This sign may be noticed in a proportion of early cases, but not in our experience before the occurrence of physical signs; moreover, it is not always easy to be certain of the X-ray appearances, and we must remember, too, that tuberculosis is not the only pathological condition which can produce such altered appearances.

An X-ray examination is, however, of assistance in certain cases. Thus in pulmonary tuberculosis associated with emphysema, in which the physical signs of the former are to some extent masked, the rays may indicate more extensive disease than had been anticipated. We have known them also to explain persistent pyrexia in a case suspected at first to be one of acute pulmonary tuberculosis, by revealing a marked enlargement of the bronchial glands, which were almost certainly infected with tubercle. Occasionally also a large, deep-seated area of tuberculous disease may be discovered by the rays before the occurrence of physical signs or the presence of bacilli in the sputum, as in the case which we shall shortly describe.

Our experience, then, is that in early cases of phthisis the diagnosis can, as a rule, be made by physical signs before the appearance of characteristic X-ray changes. Yet, seeing that cases of abnormal type do occur from time to time in which an X-ray examination is of value, the method is one which should not be neglected in a case of difficulty or doubt, *the X-ray findings being always, however, interpreted in the light of the patient's signs and symptoms.* And let us insist on the importance of the examination being in expert hands.

The following case, in which the X-rays proved helpful in detecting a deep-seated area of tuberculous consolidation before the occurrence of physical signs or the discovery of tubercle bacilli in the sputum, appears worth recording, though we must emphasise that the case is an exceptional one, and the character and position of the lesion most unusual:

L. P., aged twenty-three, a shop-assistant, came under the care of one of us as an out-patient at the Brompton Hospital in August, 1903, complaining of loss of weight and a feeling of weakness during the previous two months. He had no cough or expectoration. His saliva had once been streaked with blood, but there had been no true

hæmoptysis. His temperature, taken at the time of his visit, was 98·4°; his weight, 10 stone 6½ pounds. An examination of the chest revealed no abnormal physical signs, and the case was regarded as probably one of debility only, though his aspect and slender build gave cause for some anxiety. During the next three months his weight was maintained; his temperature was not raised, and no abnormality was revealed by physical examination. A little phlegm was hawked up, but no bacilli could be found in it.

On November 30 the weight was 10 stone 2½ pounds, a loss of 3½ pounds during the preceding fortnight. His temperature was 98°.

On December 14 slight cough and morning expectoration were complained of, but still no bacilli could be discovered. An X-ray examination was made on this date by Dr. Greg, who reported a somewhat dense shadow extending from the clavicle to the fourth rib—most dense at the centre of its area; the movements of the diaphragm on the left side were also slightly, though definitely, restricted. The appearance of the shadow is shown in the accompanying diagram (Fig. 56).

In the light of this report, the chest was again examined, but the only abnormality discovered was some cogged-wheel respiration on the left side, heard most markedly over the region of the shadow.

On February 22, 1904, his weight had risen to 10 stone 6½ pounds, and he ceased attending for a time.

On May 16 he reappeared, having kept pretty well during the interval. His weight was now 10 stone 11½ pounds. He still had a slight cough and a little phlegm. His chest on physical examination showed no new development. On June 12 the X-ray examination showed the shadow to be less dense, and less distinctly outlined, and perhaps a trifle larger. *On this date tubercle bacilli were found for the first time in the sputum.*

The patient was not seen again until October, when he was found to have gone downhill rapidly. He was still at work, but his cough was troublesome, and he brought up every morning half a teacupful of phlegm; he had had also several serious attacks of hæmoptysis. He looked thin and pallid, and his weight had fallen to 9 stone 3½ pounds. Definite physical signs were now present in the chest. The note was impaired from the left apex down to the level of the third rib, and over this area showers of fine crepitation were audible on coughing. The breath-sounds were weaker over the same region, and above the clavicle were slightly bronchial in character. On examination with the X-rays, the area of opacity was found to be a little greater and less circumscribed than when it was first observed nearly a year ago, and to have assumed the typical mottled appearance so suggestive of well-developed pulmonary tuberculosis. The patient after this date ceased attending, and we have been unable to ascertain the further progress of his disease.

The case which we have described is of interest. It was apparently one in which a comparatively large area of lung

had become the seat of tuberculosis, not in the usual situation, but in the central part of the left upper lobe, and had then undergone caseation. From its position, separated by air-containing lung from the chest wall, it failed to be recognised by physical signs; but its opacity to the X-rays produced a definite shadow some time before tubercle bacilli were found in the sputum, and before physical signs became evident. Such cases are, however, quite rare, and their occasional occurrence does not alter our conclusion that only excep-

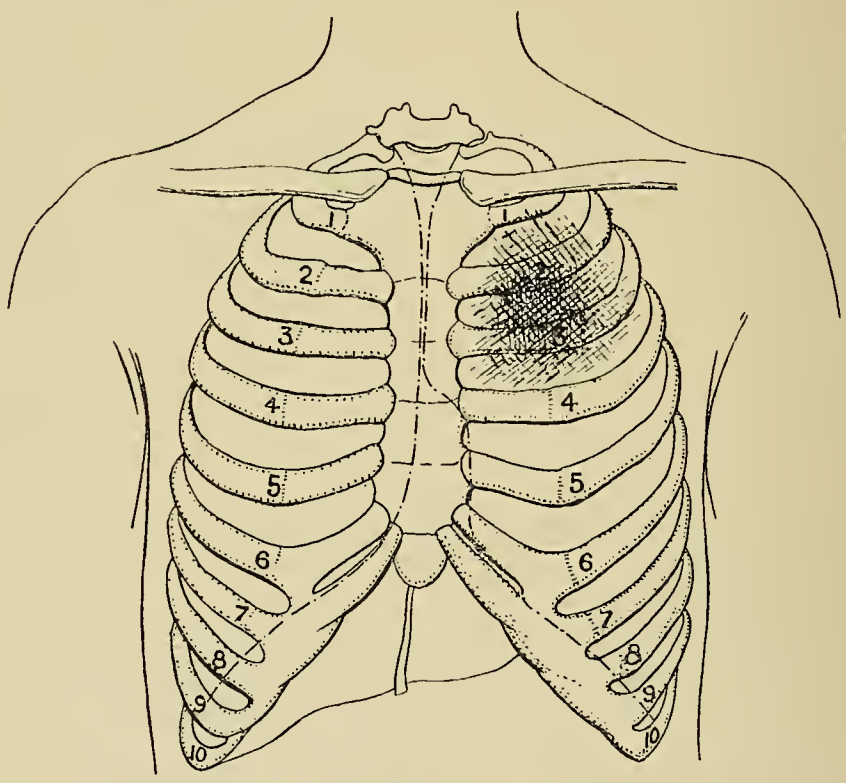

FIG. 56.—DIAGRAM SHOWING THE UNUSUAL APPEARANCES SEEN ON THE SCREEN IN A CASE OF EARLY PULMONARY TUBERCULOSIS.

tionally can we expect assistance from the X-rays in the early diagnosis of phthisis.

The Tuberculin Test.—This test depends upon the fact that a person already infected with tubercle is much more sensitive to a dose of tuberculin than is a healthy person. The test may be applied in several ways, of which we shall consider the following: (*a*) by the injection of old tuberculin under the skin in the manner originally suggested by Professor Koch (the subcutaneous test); (*b*) by its inoculation into the skin (von Pirquet's cutaneous test); (*c*) by instilling the tuberculin

into the conjunctival sac (the conjunctival test of Wolff-Eisner and Calmette).

The Subcutaneous Test.—Of the various tests, this is the oldest and the most important. It should be performed by injecting under the skin at intervals of forty-eight hours increasing quantities of Koch's old tuberculin, when, if tuberculosis is present, a reaction occurs, manifesting itself by a rise of temperature and general constitutional symptoms, accompanied often by local swelling and redness of the site of inoculation, and very possibly by some focal symptoms and signs, which serve to indicate the seat of the disease. It must be assumed that the preparation of old tuberculin is of uniform strength. It is further necessary that in all cases in which it is used the patient should be under close observation, so that any rise of temperature or other signs of reaction may be duly noted, and for this purpose the temperature should be taken every four hours for a few days prior to the injection, so that the normal range may be observed. *The test should not be applied to febrile patients whose mouth temperatures reach* 100°; a rise of temperature under such conditions would be difficult of interpretation, and the test itself might not be without risk of increasing the activity of the disease. The tuberculin is best prescribed in cubic millimetres (c.mm.), as Drs. Morland and Riviere have suggested, the cubic millimetre being the amount of tuberculin really indicated by the milligram of the older nomenclature. The doses required are supplied by the chief chemists in small sterilised phials, each of which contains the specified quantity for one injection, properly diluted for use. The doses usually given for the adult are as follows: 0·2 c.mm., 1 c.mm., 5 c.mm., and 10 c.mm. If the test remains negative after an injection of 10 c.mm., tuberculosis can be excluded. According to our experience, a reaction does not, as a rule, occur in cases of suspected phthisis until a dose of 5 or 10 c.mm. has been attained. If after an injection the physician is in doubt as to whether a reaction has occurred or not, the *dose last given should be repeated, not immediately increased.*

For children the doses of tuberculin should be smaller: 0·1 c.mm., 0·5 c.mm., 2·5 c.mm., and finally 5 c.mm.

The injections are commonly made between the shoulder-blades, as the skin is here lax, and any local swelling or red-

ness which may occur at the site of injection—and with the larger doses this is not uncommon—is but little felt. Other sites may be chosen, however, such as the skin over the lower axillary region, or, if the patient prefer it, the arm. The temperature after the injection should be taken every four hours, but unless signs of a reaction occur it is not necessary for the patient to remain in bed. It is perhaps best to give the injection towards evening, so that, if the result be positive, signs may show themselves before the patient rises next morning.

The *general reaction* manifests itself by fever, headache, pain in the back and limbs, and general feeling of malaise and illness, and differs much in degree in different subjects. If the fever does not exceed 100·5°, the reaction is sometimes spoken of as "mild"; a "moderate" or "severe" reaction being denoted by a maximum temperature between 100·5° and 102°, and above 102° respectively. As a rule, the higher the fever, the more marked are the constitutional symptoms. The temperature generally commences to rise some eight to twelve hours after the injection, though in some cases the interval may be less (Fig. 60), whilst in others the reaction may be delayed until the following day. The rise is usually rapid to the maximum, the fall more gradual, the normal temperature being reached after twenty-four or forty-eight hours, or in some cases even later.

The following charts, taken from patients under our care, demonstrate the varying temperature curves which may be observed:

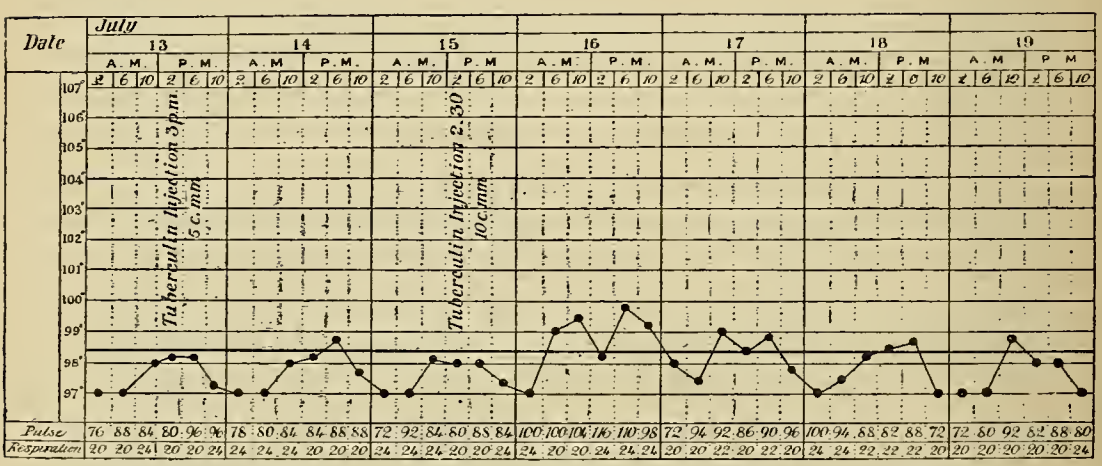

FIG. 57.—SHOWING A "MILD" REACTION PRODUCED BY AN INJECTION OF 10 C.MM. OLD TUBERCULIN.

The temperature remained a little raised for four days before returning finally to normal.

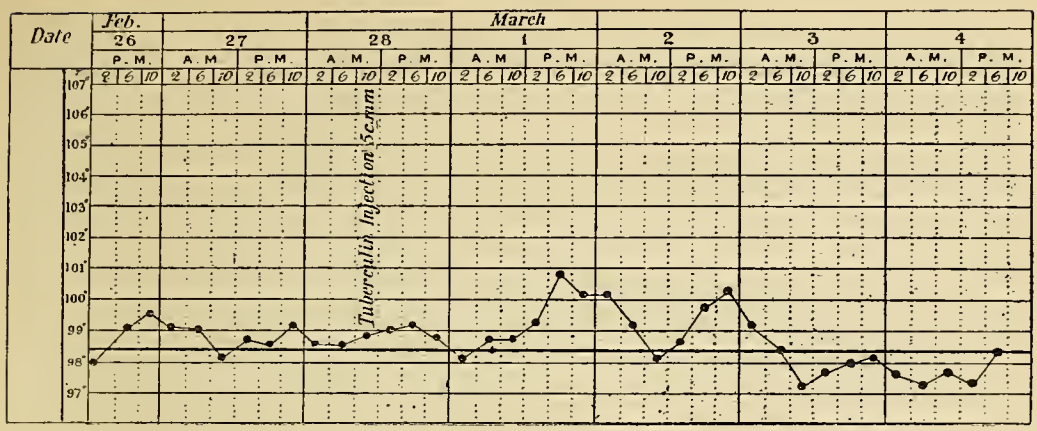

FIG. 58.—SHOWING A "MODERATE" REACTION ACCOMPANIED BY INCREASED COUGH AND PHLEGM, PRODUCED BY AN INJECTION OF 5 C.MM. OLD TUBERCULIN.

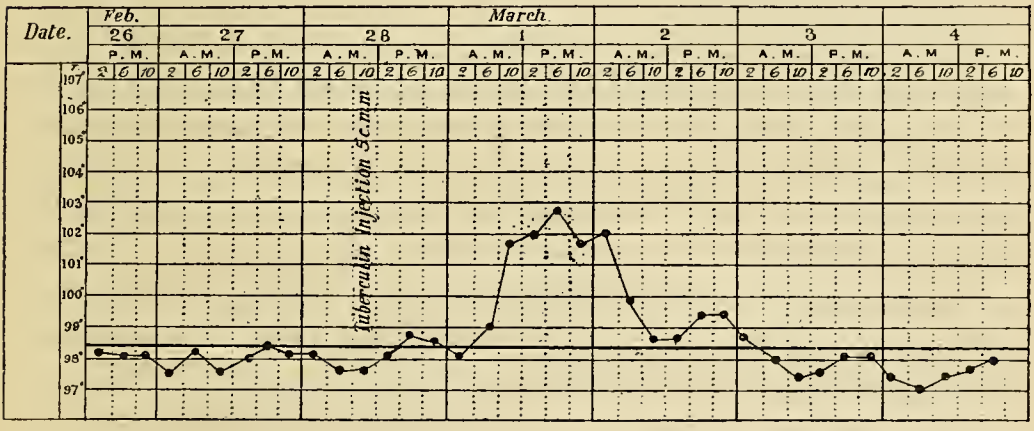

FIG. 59.—SHOWING A "SEVERE" REACTION ACCOMPANIED BY INCREASED COUGH AND PAIN OVER THE UPPER PORTION OF BOTH LUNGS, PRODUCED BY AN INJECTION OF 5 C.MM. OLD TUBERCULIN.

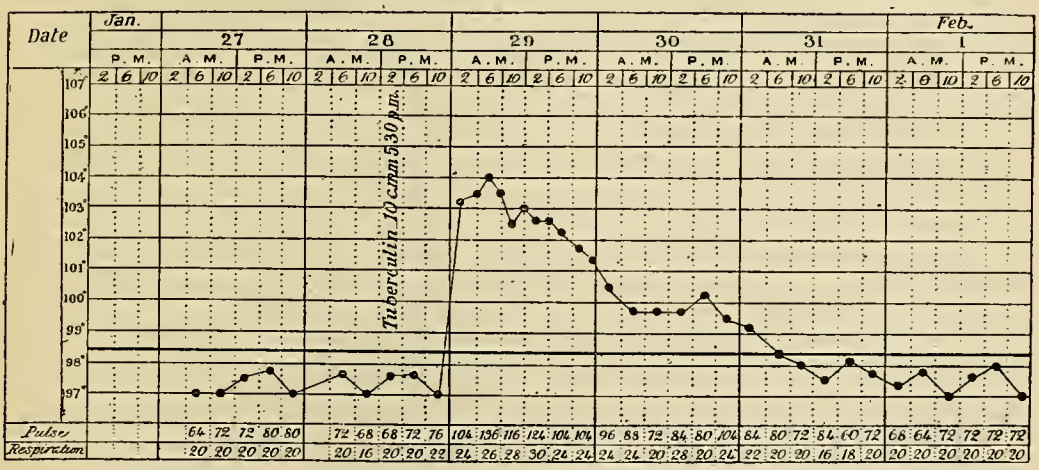

FIG. 60.—SHOWING AN UNUSUALLY SEVERE REACTION, PRODUCED BY AN INJECTION OF 10 C.MM. OLD TUBERCULIN.

We may add that no rise of temperature should be considered as a reaction unless it amounts to at least 1° F. above the highest temperature recorded during the days immediately preceding the injection. Moreover, slight rises, unless accompanied by constitutional symptoms, should be regarded with suspicion, and the matter decided by a further injection of tuberculin, the quantity last given being repeated before proceeding to the higher doses in the scale.

A definite reaction to tuberculin such as we have described indicates almost with certainty a tuberculous focus within the body. A negative reaction to the test, on the other hand, unless the patient be in the last stages of phthisis, and thus unable to react—when, we may add, the test should not be applied—is very strong evidence against the tuberculous nature of the disease.

The general reaction, however, with which we have been dealing, does not do more than demonstrate the existence of a tuberculous focus somewhere in the body. It does not prove that it is in the lung, nor does it, as a matter of fact, prove that the focus is in a condition of activity, seeing that the observations of Madison,[3] Franz,[4] Max Beck,[5] and others prove that, at least among the working classes, latent lesions may be revealed by the test in from 40 to 61 per cent. of individuals who show no sign or symptom of tuberculosis. But should the patient have been suffering from cough, wasting, or other symptom suggestive of pulmonary tuberculosis, with some indefinite signs in the lung, it is not unreasonable to suppose that the focus is situated actually in the lung itself, and that the lesion is an active one. This assumption is rendered almost certain if, as not infrequently happens with the more severe reactions at any rate, signs of a *focal reaction* in the lung make their appearance. These are manifested by pain over the chest, increased cough, and possibly by the expectoration of a little phlegm, in which tubercle bacilli may make their appearance for the first time. On auscultating the chest, crepitations are sometimes also heard at a spot where before the signs had been indefinite. These focal signs are due to the congestion and inflammatory reaction which are well known to occur at the seat of the tuberculous disease as the result of the injection of tuberculin, and which have been demonstrated so frequently in cases of lupus.

The occurrence of this focal reaction is thus of great importance, for, should it accompany the general reaction, it proves the existence of a tuberculous lesion in the lung itself, and enables the physician to recommend without hesitation important measures of treatment, of the necessity of which he may up till then have been in doubt. In other cases a negative result to the subcutaneous test has enabled us to exclude the presence of tubercle, and thus allay anxiety and avoid unnecessary expense.

The tuberculin test should not, as we have said, be used in febrile cases; nor, we may add, in cases in which there has been recent hæmoptysis, owing to the increased local congestion which results. It should be withheld also in patients with kidney disease, in those suffering from epilepsy, and those greatly debilitated from whatever cause. Mild laryngeal disease is no contra-indication to its use. If care be exercised in these respects, and in the dosage and administration of the tuberculin, we believe that the test may be safely employed, and in our own experience we have never observed harm to arise from it. *It should not, however, be used, save in those cases in which more light in diagnosis is for special reasons urgently needed.*

The Cutaneous and Conjunctival Test.—In the last edition of this work we considered in some detail the cutaneous and conjunctival tests, with which the names of von Pirquet and Calmette are usually associated. Experience has, however, shown that the cutaneous test is too delicate, responding to latent lesions, and thus commonly giving a positive result in healthy persons other than infants and very young children. It is therefore of no value in the diagnosis of phthisis. The conjunctival test, besides being of very uncertain value,[6] is apt to set up serious conjunctival trouble, and is not therefore to be recommended.

The Opsonin Test.—This method of diagnosis was introduced by Sir A. E. Wright,[7] and is based upon the estimation of the relative quantity of opsonin present in the serum of the suspected person, and a comparison of the amount with that observed in healthy individuals. Into the technique of the test we do not propose to enter farther than to say that it consists in bringing into contact measured quantities of the serum of the patient, white blood-corpuscles from a healthy person,

and an emulsion of tubercle bacilli, and allowing them to remain in the incubator at 37° C. for a quarter of an hour. At the end of this period a film is made, which is stained with carbol-fuchsin, decolourised with acid, and then counterstained with methylene blue, when the number of bacilli taken up by the leucocytes is counted. A similar procedure is then carried out, using the serum of a healthy person (or a mixed serum from several healthy people) as a control, and the number of bacilli within the cells again enumerated. The figure obtained in the case of the patient's serum, divided by that obtained when using the control or healthy serum, constitutes the "opsonic index," and gives the measure of the quantity of opsonin present in the serum, which, it is thought, so affects the bacilli as to enable them to be absorbed with readiness by the white corpuscles.

It has been shown by Professor Bulloch and others that the index among healthy persons varies between 0·8 and 1·2, and that consequently any figures within these limits must be regarded as coming within the normal. It has also been demonstrated that an injection of tuberculin produces a marked variation in the index, causing a rise or fall, according as a positive or negative phase has been produced. Turning to pulmonary tuberculosis, the work of Dr. Inman and others has shown that in active disease, even when the patient is at rest, the index often varies markedly from day to day, swinging on either side far beyond the normal. The patient is presumably absorbing from his focus of disease irregular quantities of tuberculin, and is living in a succession of positive and negative phases, that is to say, of depression or exaltation of the opsonic index. This variation of the index is well seen in the following chart (Fig. 61), from a patient in the Brompton Hospital suffering from active pulmonary tuberculosis, observed by Dr. Inman, which we have taken from his Weber-Parkes Prize Essay for 1909.

Such cases of active disease are usually, however, already diagnosed on other grounds, and these spontaneous changes in the index when at rest become, therefore, of comparatively little value for purposes of diagnosis. In early stages of the disease, on the other hand, the opsonic index is much more stable. In a certain proportion of cases of phthisis, as in other varieties of local tuberculosis, the index may be low,

and if sufficiently below the limits of health this fact may become of diagnostic importance; but in other patients the index under ordinary conditions may be within the limits of health. Under such circumstances, however, Dr. Inman has shown that active exercise, such as hard walking for an hour or manual labour, often produces a variation in the index, a fall or rise beyond the normal, owing, very probably, to an auto-inoculation of tuberculin from the focus of disease. In healthy persons no such variation is produced. These facts

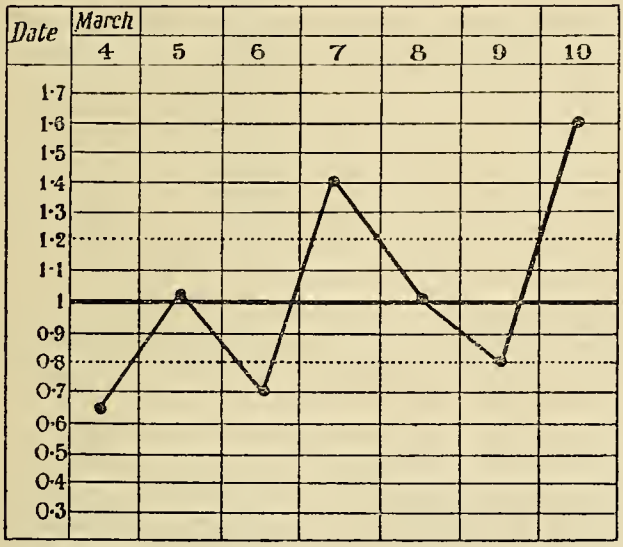

FIG. 61.—CHART SHOWING THE VARIATIONS IN THE OPSONIC INDEX OF A PATIENT SUFFERING FROM ACTIVE PULMONARY TUBERCULOSIS WHEN AT REST IN BED (DR. INMAN).

are well shown in the accompanying charts (Figs. 62 and 63) from patients at the Brompton Hospital observed by Dr. Inman, and taken from his Weber-Parkes Prize Essay.

Variations in the index following exercise such as we have been describing are commonly observed in cases of early phthisis, and when they occur, as they often do, in suspected cases, they may be regarded as evidence very suggestive of the presence of a tuberculous focus, a focus, moreover, not *latent* like those revealed in many cases by the tuberculin test, but one which is active, and from which toxines are being absorbed, and which therefore requires treatment. This conclusion has received support by carefully following up certain of the patients at the Brompton Hospital who had given suspicious physical signs, and who had yielded a positive result

to the opsonin test. In twenty of these patients repeated examination of such sputum as could be obtained was made, and in nine of them tubercle bacilli were eventually discovered before they left the hospital.

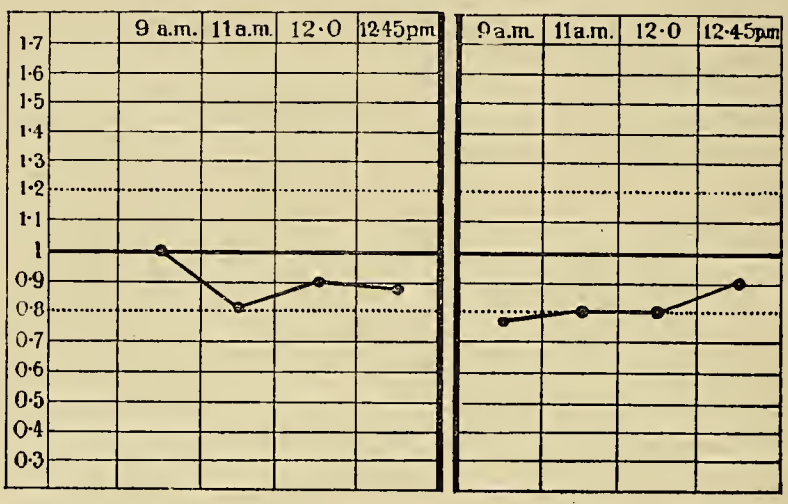

FIG. 62.—SHOWING THAT IN HEALTHY PEOPLE ACTIVE EXERCISE PRODUCES NO VARIATION IN THE OPSONIC INDEX (DR. INMAN).

Between 11 a.m. and 12 an hour's hard walking exercise was taken.

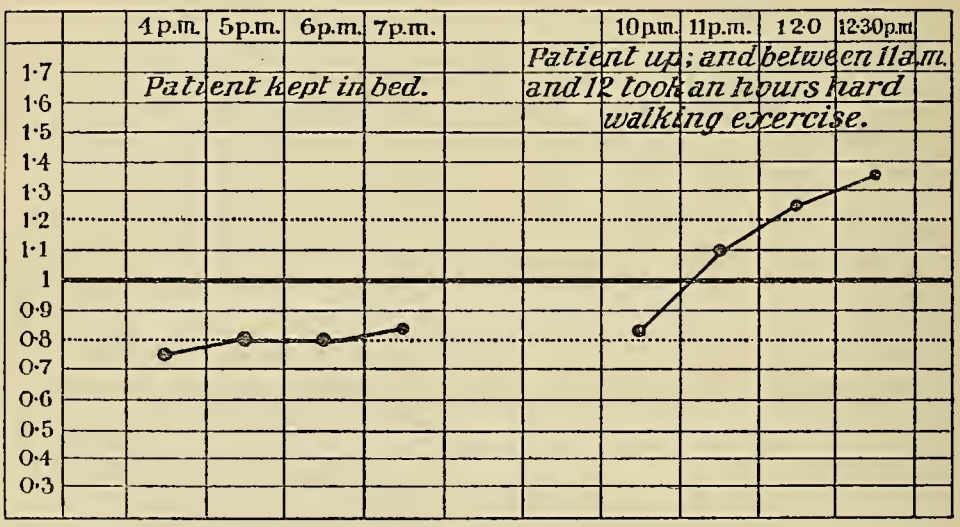

FIG. 63.—SHOWING, IN THE CASE OF A PATIENT SUFFERING FROM EARLY PULMONARY TUBERCULOSIS, AN INDEX WITHIN THE NORMAL SO LONG AS THE PATIENT REMAINED AT REST, BUT MARKED ABNORMAL VARIATION PRODUCED BY EXERCISE (DR. INMAN).

Other evidence of the reliability of the test is afforded by the fact that, as Dr. Inman has shown, and as we have pointed out elsewhere, the variations in index produced by exercise

may be observed to cease when, as the result of treatment, complete arrest of the disease has been attained.

We have now to ask ourselves how far the opsonin test which we have been discussing, and which we believe may be relied upon in the hands of observers well accustomed to its use, is fitted, by reason of the difficulties of its technique and other causes, for general adoption. In this connection we must draw attention to some interesting observations by Dr. E. C. Hort,[8] who sent to various pathologists of proved experience specimens of blood *drawn at the same time* from certain patients, and then compared the indices obtained. These showed in many cases great variation. To take one example, that of a patient suffering from pulmonary tuberculosis with well-marked physical signs, and with tubercle bacilli in the sputum, the opsonic indices returned by three observers were as follows: (*a*) = 0·88; (*b*) = 1·17 and 1·34 (two specimens of the same blood); (*c*) = 2·34. Two observations were thus within normal limits; the other two were abnormal, and pointed to a diagnosis of tuberculosis. The difference between the highest and the lowest indices amounted to as much as 1·46. We have seen the same discrepancy in observations from the same specimen of blood made by three observers in a case in which there was no tuberculosis present. In view of such results, we can only conclude that, though the method appears valuable in the hands of a very limited body of experts, it is not suitable for general employment.

It would be interesting to ascertain the relative phagocytic power in groups of persons reputed to be more or less susceptible to tuberculosis, such as town dwellers, country dwellers, those living under conditions of overcrowding in various degrees, alcoholics, feeble-minded, epileptics, diabetics and the insane, as giving insight as to the mechanism of their proclivity to the disease.

Other Specific Tests.—We may now briefly refer to certain other specific tests for the presence of tuberculosis, which, like the opsonic test, depend upon the detection in the patient's blood of various specific substances produced in response to the invasion of the tissues by the tubercle bacillus. They are of great scientific interest, but cannot be said to be as yet of much practical importance. Among them we may mention:

(*a*) *The Complement-Fixation Test.*—This test, in the words

of Dr. Inman, depends upon the fact that if a foreign albumin be introduced into an animal, specific antibodies are produced and can be demonstrated in the blood. Bacteria are such foreign albumins, and so are red blood-corpuscles. It is due to this latter fact that the so-called complement-fixation test has been made possible.

Solution of red cells may be produced by bringing them into contact with the serum of an animal previously injected with the same type of red cells, so long as a sufficient amount of complement, a substance normally present in blood, is added. In the process union takes place between complement, red-cell-antibody and red cell. The mixture is known as a "Hæmolytic system."

If tubercle bacilli are brought into contact with a serum containing antibodies to the tubercle bacillus, and a sufficient amount of complement is added, union takes place between the complement, tubercle antibodies and tubercle bacilli.

The complement-fixation test consists in bringing together tubercle bacilli, the patient's serum and complement. After a suitable lapse of time, to allow of union taking place in the event of tubercle antibodies being present in the patient's serum, red cells and red-cell-antibodies are added. In those tubes containing serum which possesses tubercle antibodies no hæmolysis can take place, for the necessary complement is already "fixed" in the combination—complement, tubercle anti-body and tubercle bacillus. The reaction is then said to be positive.

The test has, however, not proved of much value in practice, since as Dr. Inman,[9] Dr. Radcliffe,[10] and others have shown, it is negative in a certain proportion of early cases of phthisis, and as the late Dr. Meek[11] has proved, it may remain positive in patients with arrested disease who have been free from symptoms and in excellent health for several years, showing that a positive reaction does not necessarily indicate active disease.

(*b*) The *Precipitin Test* and (*c*) the *Cobra Venom Test*,[12] which we described in our last edition, possess also a scientific rather than a practical value.

(*d*) The *Agglutination Test*, which has been found so valuable in typhoid fever, was applied to tuberculosis by Professors Arloing and Courmont[13] some years ago, and they have re-

corded several successful results in a series of papers dealing with this subject. In other hands, including our own,[14] the method has not been found to give reliable results in practice, and we cannot therefore at present recommend it. If, however, the difficulties in obtaining a culture yielding a satisfactory emulsion of tubercle bacilli can be overcome, the possibilities of the test are great.

Although these tests must for the present be regarded as mainly of laboratory interest, they are nevertheless of value in helping to deepen our insight into the intimate pathology of the disease and the *modus* of its immunity, and probably in furnishing some clue to its prophylaxis and treatment in the future.

Conclusion.—We may, in conclusion, sum up the main facts with which we have dealt in this chapter, and indicate the procedure which we should adopt in arriving at a diagnosis in any doubtful case of pulmonary tuberculosis.

The aspect of the patient is important, and careful inquiry should be made into the history of the illness, including such symptoms as cough, loss of weight, and sweating. The existence of pyrexia is of great moment, and its presence or absence must be definitely decided by a night and morning record of the temperature for a period of a week or more, if necessary. The chest should be carefully examined, and great stress must be attached to abnormal physical signs, such as slight impairment of note at one apex and weak or harsh breathing, with or without a few moist sounds after cough, signs which are commonly observed quite early in the disease. Speaking from our own experience, we may say that a history such as we have indicated, if accompanied by slight pyrexia and by abnormal apical signs, is almost diagnostic of pulmonary tuberculosis, and the examination of the sputum but confirms the conclusion.

Nevertheless, in all cases the expectoration should be examined, and the diagnosis, upon which far-reaching and expensive methods of treatment may depend, placed, if possible, beyond doubt. For this purpose it is best to have the early-morning sputum tested for tubercle bacilli by ordinary methods of staining on three successive days; if the result be negative, the antiformin or some other concentration method should then be employed. In most cases of this kind,

even if it be stated that there is no sputum, a little can be obtained if the patient is directed to carefully collect any particles which he may cough up in the early morning on first waking, aided if need be by a few doses of an expectorant mixture. In children who swallow all sputum it is sometimes possible to detect the bacilli in the stools, which in such cases should be examined by the antiformin method. We have known this test prove successful.

Should it not be possible to demonstrate the presence of tubercle bacilli, even after the most rigorous search, and the diagnosis remains seriously in doubt, further tests may be employed. In some cases help may be derived from the X-rays; but as a rule in early cases of phthisis too much must not be expected from this method of examination, which is by no means free from fallacies.

Of some assistance is a determination by a skilled observer of the opsonic index before and after exercise, when in many cases of tuberculosis an excursion beyond the normal can be obtained. But, for reasons which we have given, the method is not suitable for general employment, and in all cases the results obtained must be most carefully considered in the light of clinical observation.

In afebrile cases, when the need for a more certain diagnosis is pressing, the subcutaneous tuberculin test may be employed, and for such cases it should be reserved. The occurrence of a general and constitutional reaction indicates a tuberculous lesion somewhere in the body, but not necessarily an active one; signs of focal reaction in the lung, which are alone diagnostic, and which must be carefully watched for, point to pulmonary involvement. In the cases which we are considering a negative result to the test would exclude the diagnosis of pulmonary tuberculosis.

Von Pirquet's cutaneous reaction is too delicate to be of value; and the conjunctival test is not to be recommended.

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CHAPTER XLII

GENERAL OBSERVATIONS ON THE PROPHYLAXIS AND TREATMENT OF PULMONARY TUBERCULOSIS

THE prophylaxis of pulmonary tuberculosis, generally speaking, does not essentially differ from that of other diseases; all measures that tend to increase constitutional vigour also diminish mortality from consumption. On glancing through the chapters on Ætiology, however, it will be seen that there are certain conditions, specific and other, which especially favour the occurrence of tuberculosis more than of other diseases, and that the tendency to the disease is inherited in a certain proportion of cases. By amending those conditions, especially overcrowding, sedentary employment, dusty occupations and intemperance, which favour the incidence of phthisis, and by taking extra precautions in all cases of hereditary predisposition, we do the best that can be done, generally speaking, in the way of prophylaxis.

There are those amongst physicians and sanitarians who are still disposed to minimise hereditary influence in phthisis, but without doubt, in our opinion, it should be taken into account in considering the question of marriage. There are cases in which the disease has manifested itself so strongly in both the contracting families as to make marriage absolutely undesirable. Other less decided cases must be considered on their respective merits.

General Precautions.

The child of a consumptive mother should be weaned early, and should, when possible, have a carefully selected wet-nurse. If a healthy foster-mother be provided, the child should be suckled entirely until eight or nine months old. If the alternative of a wet nurse be not adopted, careful hand-feeding by

humanised or other artificially prepared milk must be employed, attention being paid to an adequate provision of the requisite vitamines.

The hygiene of the nursery must be strictly looked to, especially with regard to the avoidance of dust, the free admission of air without draughts, the provision of a separate cot or bed for the child, and abundance of air-space in the night nursery. Milk, which should be boiled or properly pasteurised, must form a large item of the dietary throughout childhood, the other elements—saccharine, farinaceous, nitrogenous and saline—being duly provided. Careful attention to the skin, by the use of a tepid bath at least once a day, warm, light all-wool underclothing, loosely fitting and made to cover the chest to above the clavicles, and abundant outdoor exercise, are the principal means of securing sound health. The plan of keeping delicate young children with naked legs, arms, and upper chest, with the view of hardening them, is too obviously absurd to need further comment. It is, perhaps, at the present time unnecessary to add that the nurse herself must be healthy and strong, and free from any suspicion of tuberculous disease.

At the period, about the age of eight, when children begin to go to school, the question comes whether a day-school or boarding-school shall be selected; and in most instances it is decidedly preferable that delicate boys, and perhaps girls, should be sent away from home to a carefully-managed preparatory school, than that they should attend a day-school from their own homes. The divided responsibility in matters of hygiene, food, clothing and the like, the hurried meals, irregularity in exercise, and increased exposure to cold and wet in bad weather, which attendance at day-schools involves, are full of risks. There is also no doubt that delicate children, from the extra solicitude which has been necessary in rearing them, become whimsical and morbidly self-conscious at about this age, and the regularity and discipline of school life is good for them. The school should be favourably situated on a well-drained soil in the country, at the seaside, or in the neighbourhood of the sea. The modified sea climate of Bournemouth suits children well, also the high ground of St. Leonards, Ramsgate, Broadstairs, Folkestone, and many other places. The experiment of sending delicate children to the Swiss mountain resorts is of doubtful expediency. It can only be

prudently tried in cases where abundant means will permit of the best conditions and supervision.

Whooping-cough and measles are antecedent to a large proportion of cases of tuberculosis in children, and the utmost care should be taken to insure complete convalescence from these diseases.

An undue irritability of the lymphatic glandular system is often to be observed in children prone to phthisis, and the glands, when enlarged, not infrequently undergo caseation, and remain, as it were, magazines of tuberculous poison. Hence all sources of gland irritation, decayed teeth, eczemas, eruptions on the scalp, catarrhal affections of the bowels and bronchi, should be carefully and promptly treated. Enlarged tonsils and adenoids should also receive attention. Chicken-pox in children often leaves behind troublesome sores about the head and body, which are very likely to lead to glandular enlargements, and the utmost care should be taken during this disease to cleanse and protect all pustules which are large and likely to ulcerate. In cases in which the glands become caseous and suppurate it is better, if possible, to make a clean excision of the gland, or at least to remove all caseous matter by thorough scraping; less scarring is thus caused than by simple incision, and the danger of infection of other glands by retained caseous material is removed. Short courses of cod-liver oil and steel wine or Parrish's Food should be given to delicate children, extending over three or four weeks, at intervals during the winter and spring, and especially after the occasional catarrhs to which they are all liable.

There is some foundation in experience for regarding the succeeding periods of seven years as critical in matters of health: there are certainly grouped about the first and second dentition, and the periods of adolescence and manhood, developmental changes and associated external circumstances of life which favour the occurrence of certain diseases, and peculiarly of tuberculosis.

Between the ages of fifteen and twenty-one phthisis is very liable to develope in those predisposed, and an opportunity may be taken after the completion of school education, or at the end of the college career, to secure a period of six or twelve months to be devoted to the establishment of sound health. Many plans may be devised with this view, suited to the cir-

cumstances and means of the patients and their friends. A long sea voyage with a responsible companion is one of the best measures, or twelve months' residence on a farm in a healthy part of this country or one of the colonies. With girls it is more easy to arrange a series of visits to healthy parts of the United Kingdom or abroad, where an outdoor life can be, to a great extent, secured. The future profession or business of a youth may during this time be determined upon, sedentary pursuits being avoided, and those encouraged which are associated with an active outdoor life. There are some cases, however, and they are not very uncommon, in which, with tuberculous predisposition, the mental faculties are keen, whilst the bodily conformation is not such as to withstand, or respond to, a rough physical life. In such cases a sheltered life is to be recommended, with such pursuits as the individual is best qualified for, and with such precautions in the way of hygiene, exercise, and climatic change as may be possible and best adapted to the case. The experienced physician can recognise in many such cases that the fund of vitality is small, and that to attempt to lay it out on an ambitious scale, with a view to large or long-continued returns, is to risk the loss of the whole.

"Neglected colds" enter into the history of a large proportion of cases of phthisis. The best routine treatment of an acute catarrh in a delicate person during the first twenty-four hours consists of repeated doses, at short intervals, of citrate of ammonia or potash, the patient remaining in bed or in a warm room. Frequent fomentation of the nasal passages with hot water should be adopted, and a hot foot-bath and a little Dover's powder given at night. Besides hot-water fomentations, the local treatment of a nasal catarrh consists in the use of some antiseptic inhalation, spray or douche. The vapour of equal parts of the oil of eucalyptus and eau-de-Cologne may be inhaled, or the glycerine of thymol douche employed, or various antiseptic drugs. In two days quinine may be commenced, the patient still remaining indoors. After about the third or fourth day, when all febrile symptoms have subsided, a week's change to some accessible seaside place will commonly cure the catarrh. Sometimes at the first onset of the malady a few fairly full doses of quinine will arrest it; a combination with ammonia in the form of the ammoniated

tincture is particularly useful. Every effort must be made to prevent a catarrh from lingering or becoming chronic. In all cases of acute inflammatory chest affections occurring in patients with tuberculous tendency the utmost care should be taken to insure complete recovery, and it often takes a long time thoroughly to effect this. It is well, if such attacks are repeated, to advise that the ensuing winter be spent in taking a voyage, or at a health resort adapted to the circumstances and case.

In patients in whom there is an inveterate tendency to recurrent catarrh, whether influenzal or otherwise, it may be desirable to ascertain the organism of infection—Bacillus catarrhalis, Pfeiffer's bacillus, the pneumococcus or other—and to prepare a vaccine to be used at appropriate intervals, with a view to increase the resistance to these organisms, and thus ward off the attack.

General Observations on Treatment.

There is much in the treatment of phthisis which is in common with the treatment of other diseases. The four following points should, however, be especially remembered with regard to pulmonary affections:

1. During respiration samples of the surrounding air are constantly being brought into contact with the extended respiratory surface, some portions of which surface are, in the cases under consideration, more or less lacerated or suppurating, and bathed in muco-purulent matters, ready to decompose, and abounding in specific germs.

The remembrance of the facts embodied in this statement is enough to emphasise most strongly the importance of all hygienic measures calculated to keep the air pure, and free from organic and inorganic dust, and the necessity of abundant cubic space being allotted to such invalids, even beyond the requirements of others.

2. All the blood of the body passes through the lungs, the pulmonary circulation in this respect balancing the systemic. From this fact flow two or three considerations in the treatment of phthisis.

(*a*) Any conditions which hurry the general circulation cause an unduly proportionate stress of blood-current through

the lungs, and hence the importance, during active disease of these organs, of muscular and mental quietude.

(*b*) In chronic pulmonary lesions in which the disease, having effected a certain measure of destruction, is stayed, and the patient is regaining strength, flesh, and colour, a point is not infrequently attained when there arises a relative systemic plethora, the blood-volume and systemic metabolism outbalancing the vascular and functional capacity of the lungs. Fresh pulmonary hæmorrhage, congestions, dyspepsias, diarrhœa, are the natural consequences which tend to rectify this perverted balance, but which, once started, rarely stop within salutary bounds. Timely moderation in tonics, and a reconsideration of the dietary and exercise, will avert such disasters.

3. The general circulation includes the local, and as the blood-current passes through the tuberculous lesions the toxines of the tubercle bacilli and other attendant organisms are taken up in greater or less proportion, according to the activity of the local processes and the flow of blood. This absorption of toxines under the influence of the accelerated blood-current during active exercise may overwhelm the resistance of the patient, raise his temperature, and prostrate his nerve-power. With perfect quiescence absorption is lessened, the index of resistance is raised, and the temperature lowered. In chronic lesions exercise may be so regulated by observation of the temperature range as to produce immunity by securing duly adjusted toxic absorption. We shall more fully discuss this important matter in the next chapter.

4. Considerable tracts of lung are in health held in reserve for temporary service on occasions of unwonted exertion. It is the development and bringing into daily action of such reserves that constitutes a most important element in arrest of, or "recovery" from, phthisis. This development can be encouraged at the fitting time by graduated exercise on the incline, by residence at high altitudes, or by both combined, perhaps also by the use of apparatus such as we have referred to when discussing "pneumatometry" (p. 25). Mere expansion of lung, however, be it remembered, does not constitute compensatory development; there must be also increase of capillary circulation and nutrition. The cautious stimulation of blood-pressure and respiratory function gained by regulated

exercise most efficiently aids the natural tendency to the changes desired.

The Hygiene of the Sick-Room.—This is a matter of the utmost importance in the treatment of phthisis. The dwelling-rooms of the patient should be of good size, lofty, and well ventilated, with a free exposure to the south or south-west, and sheltered from the north and east; for the bedroom a south-east aspect is the best, giving better morning sunlight. The windows should be kept widely open day and night. French windows are preferable, as they can be adjusted to protect against any prevailing high wind, whilst permitting free entry of air. The furniture should be sufficient for comfort, without superfluity. Carpets and curtains should be easily removable, so as to be shaken and dusted out of doors. All sweeping of carpets should be strictly prohibited in the invalid's rooms, and the floors and furniture should be kept free from dust by the use of cloths rendered damp by Sanitas fluid or other weak solution of a cleansing kind. Washing chintz coverings to the furniture are greatly to be commended, and two or three well-chosen patterns will afford a change from time to time, grateful and cheering to an invalid much confined to a suite of rooms. Every few months rooms much occupied should be thoroughly cleansed, the walls and ceilings fresh papered, or lime-whited and coloured. If papered with "Salubra" paper, they should be thoroughly washed with some disinfectant solution such as cyllin (4 ounces to a gallon of water).

Much irritation, cough, and increased activity of disease, will be avoided by these simple measures, and the more the patient is confined to his rooms, the more essential is it that strict attention should be paid to them. In the presence of broken and highly absorbent surfaces deprived of the means of rejecting harmful matters, the mechanical irritation of inert dust, and the septic influence of putrefactive and other organisms, are fertile of mischief. Gas-lighting should be forbidden in the living-rooms. The patient must have a separate bed, springy, with horsehair mattress, not curtained, and sufficiently, but not heavily, covered. In bed-ridden cases it is often a good plan to have two beds in occupation, so that a change may be made from one to the other. The clothing of the patient should be warm and light, and even in the

warmest season thin woollen or silken garments should be worn next the skin.

A reference to the chapters on Ætiology will emphasise the importance of adopting every possible measure of cleanliness with regard to the disposal of the expectoration from consumptive patients; for, directly or indirectly, this is the great source of danger. We are in the habit of advising for bedridden cases a bib kept moist by a weak formalin solution, which serves as a constant and salutary inhalant and to catch and diminish the infectivity of any spray which may result from coughing. As a further precaution, a Japanese handkerchief should be brought across the mouth when expectorating. The employment of suitable spitting vessels is to be insisted upon, and the proper disinfection of such vessels and the efficient destruction of their contents is equally important.

Spittoons of various patterns may be used, the essential point being that they contain a *liquid*, not a dry, disinfectant. Sputum is only dangerous when dry, so that some liquid in the receiver is of the first importance, the exact nature of the liquid being comparatively immaterial—Sanitas or weak carbolised solutions may be used; cyllin (Jeyes' fluid), 1 in 400, is also effective, and not expensive.

The next point of importance is the cleansing of these vessels and the destruction of their contents. Destruction by fire is one of the best and easiest ways of dealing with the sputum, which in private cases, when the amount is quite small, can be simply poured on to the fire; or a spitting-cup may be used, consisting of a metal frame, into which fits a stiff and water-tight paper case shaped like a spittoon, capable of holding a disinfectant; this, after use, can be lifted out of the frame, burnt, and replaced by a fresh one. When larger in amount, so that there may be a possible risk of the sputum falling unburnt through the fire into the grate beneath, and here becoming dried and a source of danger, it is best to pour the sputum, mixed with the disinfectant, into the water-closet and thus into the drainage system.

In hospitals and sanatoria, where the amount of sputum to be dealt with is greater, special methods have to be employed. At the Brompton Hospital a cremator was erected, and for some years the sputum was destroyed in this manner. The method was effective, but the cremator was found to need such

constant repair that another method had to be devised. That now in use consists in subjecting the sputum to superheated steam (250° F.) for a period of twenty minutes in an apparatus devised for the purpose. After sterilisation, the steam is turned off, and the disinfected sputum is allowed to cool down nearly to 100° F., when, by opening a valve, it is allowed to pass by gravity into the drain. This apparatus, devised by Dr. Paterson, at the time resident medical officer, and Mr. Kirkland, consulting engineer to the hospital, has now been in use for some years, and is found to work simply and well.[1] A duplicate, installed at the King Edward VII. Sanatorium, Midhurst, has given equally good results.

Glass, china or metal spitting vessels, when emptied, must be scalded out, and cleansed with washing-soda and water. Consumptive patients, when out of doors or travelling, should carry suitable pocket flask spittoons. The habit of expectorating into handkerchiefs should be avoided as much as possible, and handkerchiefs so used should be frequently changed, and at once thrown into some convenient receptacle containing a disinfecting fluid, then scalded and sent to the wash. For bed-ridden patients an abundant supply of small squares of linen, butter-muslin or Japanese paper should be at hand on a vulcanite tray or glass slab, and should be burned when used. All these precautions, it must be especially remembered, are sanitary measures, to be adopted in the interest of the patient himself, who is the most susceptible to unhygienic surroundings, as well as of others in his neighbourhood. With such measures of careful hygienic cleanliness, which can be easily carried out without fuss or ostentation, any anxiety as to the contagiousness of the disease may be allayed.

Dietary.—The dietary of the phthisical patient is a very important question, which we shall consider in detail in a later chapter. We may here, however, say that it must be framed on a liberal scale, so as to contain a due share of animal and vegetable food and salts. The appetite and digestive powers of the patient are in many cases sufficient guides as to the amount of food to be taken. The results of Debove's method of feeding phthisical patients artificially by means of an œsophageal tube showed, however, many years ago, what clinical observation also teaches, that appetite often fails when the system is, nevertheless, ready and able to assimilate much

larger quantities of food. This fact is especially to be recollected during the hectic period of phthisis, when it is most important to sustain the patient by nourishment, given in much larger quantities than inclination would prompt him to call for, and yet without resorting to that forced dietary which was formerly in vogue, and which is now justly discredited.

In cases of quiescent phthisis the natural appetite returns, and patients as a rule take food with avidity. Sometimes, when flesh and blood are being rapidly regenerated, and a tendency is observed for the body-weight to pass beyond the lung capacity, it is advisable to restrict the dietary somewhat, by diminishing malt liquors, substituting fish for butcher's meat, and suggesting some restrictions in the amount of fluids and solids taken. Coated tongue, quickened pulse and respiration, restlessness, dyspepsia, and increased breathlessness on effort, will, in the absence of any fresh lesion, lead to the recognition of this condition, already referred to as one of *relative plethora.*

The digestive system of tuberculous patients requires careful attention, and in many cases treatment mainly consists in establishing a working equilibrium between the digestive powers on the one hand, and the quantity and quality of food taken on the other, the aid of medicines being called in to support digestive powers and to correct digestive failure. A fertile source of dyspepsia is the swallowing of expectoration, and patients should be carefully warned of this.

From time to time certain special kinds of diet have been suggested as curative of phthisis, such as the *milk cure*, the *whey cure*, *koumiss treatment*, the *grape cure*, and the like. None of these measures of treatment will, as "cures," bear examination, much of the benefit being attributable to the healthy surroundings of the "cure," and it is now admitted that such cures are only adapted to a limited number of favourable cases of the disease. This limited number of favourable cases of phthisis, it must, indeed, be confessed, make the reputation of every health resort and "cure" in turn, and attract to them many other cases for which they are not suited.

The exclusive use of *milk* is not adapted for the treatment of any form of consumption, but in all cases, and especially in young subjects, milk to the extent of from one to two pints,

boiled or pasteurised, may be taken daily by those who can digest it until the body-weight is a little above the normal height-weight ratio. Cow's milk (undiluted or skimmed), ass's milk, goat's, mare's, or fermented mare's milk (koumiss), may be used. In some cases whey may be preferred; its nutritive value is not large, but for those who cannot take other forms of milk it may be tried, especially in cases in which there is a considerable loss of salts in night perspirations.

Koumiss, the fermented milk of mares, has from all time been used as a beverage by the inhabitants of the steppes of Southern Russia, the best koumiss being prepared from pasture-fed mares which have not been put to work. Ssamara is the steppe district where Russian koumiss of the best kind is made, and the best quality is obtained in May, June and July, when the climate, clear, dry, and aromatic, is said to be very beneficial. A glass or two is taken in the early morning, three or four glasses in the forenoon, and as many in the afternoon. No other drink should be taken, and no sweets or alcohol in any other form, the meals consisting of a liberal allowance of mutton, poultry, eggs, butter and bread. The treatment must be commenced with caution, and should extend over a period of two or three months, as much time as possible being spent in the open air, riding or walking.

We fear it will be some years before Russia can be visited for purposes of health, and the koumiss cure is not therefore likely to attract at the present time many patients from this country; nevertheless, the beverage is one which will be found useful on occasions, certain patients retaining it when the stomach will tolerate nothing else. True koumiss made from mare's milk is difficult to obtain in London, but a substitute prepared from cow's milk is supplied by various dairies. The following formula, kindly supplied to us by the late Dr. Charles, of Cannes, may be found useful:

"*Home-made Koumiss.*—Fresh milk to be just boiled, then, when nearly cold, put into champagne quart bottles, leaving enough room to shake it up easily; add a teaspoonful of crushed lump sugar, and a piece of German yeast* about the size of a hazel-nut—*i.e.*, 20 grains; cork with new corks, and tie down with wire or string; keep in a cool

* The German yeast can be obtained from any baker who makes Vienna or fancy bread. It soon putrefies, and should therefore be used fresh; but if placed in a cup loosely covered up with paper, it will keep a week.

place, lying down, and shake twice a day. The koumiss will be ready to drink on the sixth day in average weather, earlier in hot, and later in cold, weather. A thinner koumiss is made from skimmed milk. This more resembles the koumiss made from mare's milk. Most people can digest that made from unskimmed, and *for them* it is a mistake to use skimmed milk. All the bottles, corks, etc., must be scrupulously clean."

The Grape Cure.—This is especially carried on at Meran, Botzen, Montreux, and some other resorts in Europe, during September and October. Professor Lebert[2] recommended half a pound of grapes to be taken early in the morning, at 7 a.m., and again at 5 p.m., and, after a few days, a third quantity at 11 a.m.; following this, if the fruit be well borne, the total quantity taken each day may be gradually increased to 2 pounds, this being the safe limit to which this treatment can be carried in phthisis. The diet at other meals must be light, digestible, and unstimulating. There is no doubt that cases of phthisis are frequently not allowed sufficient vegetable food and salts in their dietary, and when and where grapes are in season the substitution of them for the intermediate meals may often be of value, especially in cases of hectic associated with torpidity of the bowels.

Exercise.—This must be taken or withheld in accordance with the patient's strength and the activity and stage of the disease. In the active phases of the malady, with elevation of temperature, quick pulse, and hurried breathing, all symptoms will be intensified by exercise, and complete muscular rest must be enjoined, in association with the best air conditions that can be provided. In connection with the digestion of food, rest is of great importance, and in many cases of phthisis we are in the habit of enjoining as a minimum of rest one hour reclining after breakfast, one before and one after luncheon, and a fourth from six to seven, the patient "being about" and taking such exercise at other times as may be advised. By means of revolving shelters, tents, sheds, bath-chairs, movable beds,* and appropriate arrangement of wraps, all the advantages of open air may be obtained without exercise in suitable climates and seasons; and much care in room hygiene on the lines already laid down will compensate for extra time spent indoors in consequence of bad weather, or in advanced

* One of Ward's, Aldermann's, or Carter's mechanical bed-chairs is a great luxury as an addition to the sick-room furniture.

cases. In early stages of quiescent cases an outdoor life is to be advised, and those occupations and climates selected in which this can be best attained. For further details we must refer the reader to the chapters on Climatic Change and Sanatorium Treatment.

REFERENCES.

[1] For a full description of the Apparatus see "The Sterilisation of Tuberculous Sputum and Articles infected by the Tubercle Bacillus," by Thomas Kirkland, M.I.C.E., and Marcus S. Paterson, M.B., *The Lancet,* 1906, vol. ii., p. 426.

[2] Quoted by Dr. Burney Yeo in the chapter on "The Grape Cure" in his work on *Climate and Health Resorts,* p. 313. See also *Climatotherapy and Balneotherapy,* by Sir Hermann Weber, M.D., and F. Parkes Weber, M.D , p. 621. London, 1907.

CHAPTER XLIII

TREATMENT OF PULMONARY TUBERCULOSIS IN ITS EARLY STAGES

Sanatorium Treatment.

TUBERCULOSIS of the lungs may, as we have indicated, manifest itself as an acute form of the disease, associated with high fever, and marked by rapid spread of the tuberculous process. This variety must be treated by absolute rest in bed on fresh-air lines, the strength being maintained by an abundant dietary, and the special symptoms relieved by appropriate medicines (Chapter XLVII.). Such cases are, however, not now before us, and we have in this chapter to consider the lines which should be adopted in the more ordinary cases in which the onset of the disease is insidious, marked, perhaps, by cough, wasting, or hæmoptysis, in which the process is less active, and fever, if present, is only slight in degree.

With regard to such cases, the first essential is to place the patients under the best hygienic conditions obtainable, and to secure for them an abundance of the purest air. The diet must be ample and nutritious, not in great excess, but duly proportioned to the body-weight. As convalescence becomes established, gradually increasing exercise should be prescribed under careful observation. Such treatment may be carried out in a patient's own home, if it be satisfactorily situated, and a carefully-trained nurse be available. Medical practitioners are now becoming in increasing numbers sufficiently well versed in the management of these cases. It is not often, however, that the home conditions are entirely satisfactory, and we have no hesitation in saying that in the majority of instances it is best for the patient to go for a time to a sana-

torium, where he will receive the individual attention which his case requires, and where he will be educated in the life which he must live after returning home.

It is not our purpose here to describe in detail the construction of a sanatorium. Such institutions, foreshadowed by George Bodington[1] in 1840, and Henry McCormac[2] in 1855, and brought into practical operation by Brehmer at Görbersdorf, and Dettweiler at Falkenstein, are now to be found in greater or less numbers in most civilised countries. They consist essentially of buildings specially designed to enable the patient, when within doors, whether in a bedroom, dining or recreation room, to live almost as it were in the open air. To this end the patients' rooms are built facing towards the south; the rooms are large and airy, and the window-space of unusual size. A veranda in front, on to which the windows open, and on which the patient may lie out during the rest-hour, is an advantage. By the construction of an open corridor to the north, into which the doors of the sleeping-room, and the windows above them, open, a continual circulation of air in the room is obtained. To avoid undue dampness, care is taken, in selecting a site for the sanatorium, to choose a dry subsoil, often in pine and heath-clad districts, but with the trees not too near the buildings. Protection from wind is usually secured by rising ground to the north, and often also by belts of trees to the north and east, which, if sufficiently dense, form an effective screen. To facilitate cleaning, and to preclude the harbouring of infected dust, all corners in such buildings should be rounded and the furniture simple, and of such a kind as may be easily wiped over with a damp cloth. Carpets should be avoided, but slips of rug may be allowed for comfort.

On first being received into such an institution, the patient should be kept in bed for about a week, so that a careful estimate of the activity of the disease may be obtained. If the temperature prove normal, the patient may then be allowed up for a short time in the day, the duration to be gradually extended, provided always that the extra exertion does not lead to any febrile reaction. Later on exercise may be permitted, beginning, perhaps, with a quarter of a mile slowly walked each day. If all goes well, this is gradually increased, and soon the patient is able to enter into the life of the institu-

tion, in which each hour is mapped out for him under careful supervision. At the King Edward VII. Sanatorium, near Midhurst, the daily programme, as set forth in the "Daily Routine and Rules for Patients," is somewhat as follows:

7.30 a.m.	Gong is sounded. Patients take their temperatures and get up. Baths, hydrotherapy, etc.
8.15 ,,	First breakfast gong.
8.30 ,,	Breakfast gong.
9-9.30 ,,	Leisure. Books can be obtained from the Librarian.
9.30 ,,	Gong is sounded. All patients must go direct to their rooms, or to that part of the balcony immediately outside their rooms, and rest on their chairs until they have been seen by their medical officers.
10-12 noon.	Rest or exercise as prescribed. On one day in each week patients will attend in the consulting-room at this hour for examination.
12 ,,	Gong is sounded. All patients must go direct to their rooms.
12-1 p.m.	All patients must rest in their rooms or on their balconies. No talking is allowed.
1 ,,	First luncheon gong.
1.15 ,,	Luncheon gong.
2-2.30 ,,	Leisure.
2.30-4 30 ,,	Rest or exercise as prescribed.
4.30 ,,	Tea in entrance-hall.
5-6 ,,	Recreation hour for music, games, etc.
6 ,,	Gong is sounded. All patients must go to their rooms as at twelve o'clock.
6-7 ,,	Rest hour.
7 ,,	First dinner gong.
7.15 ,,	Dinner gong.
8-9.30 ,,	Recreation.
9.30 ,,	All patients must go to their rooms.
10 ,,	All lights out.

With reference to the above, we would insist that the hours of "leisure" and "recreation" are not to be spent in violent amusements, which may be as tiring as any exercises prescribed. During these hours the patient's time is more his own. He is allowed to amuse himself, play chess, draughts, dominoes, and so forth, or at certain times such simple games as putting, clock-golf, or bowls. Music and singing are allowed under careful supervision, for it must be remembered that both entail muscular exertion, which, if carried too far, may do harm.

With regard to the body temperature, which patients are

rightly taught at sanatoria to take themselves, we may here remark that much discussion has been evoked as to the relative merits of mouth and rectal temperatures. The superiority of the latter has been warmly upheld by Dr. Otto Walther of Nordrach and his followers, and the rectal method is now in use at many sanatoria. There can be little doubt that the temperature so registered does more nearly represent the temperature of the body than that taken in the mouth, groin or axilla. Further, it is not subject to certain fallacies, such as the cooling of the mouth by talking, great external cold, or by a cool wind blowing upon the cheeks, by all of which the mouth temperature may be affected. Theoretically, therefore, the rectal method may be defended. It is, however, naturally repugnant to many patients of refinement, and in practice we believe that it is unnecessary, since any abnormal rise of temperature, to 99° or higher, is accompanied by subjective symptoms such as headache, loss of appetite, and other feelings of malaise. Under such circumstances the patient, if properly instructed, will take his temperature without delay and with especial care, and thus insure a correct mouth-reading. Working on these lines, satisfactory results have been obtained at the Brompton Hospital Sanatorium at Frimley, and we think that they go far to prove the adequacy of mouth temperatures.

Exercise and Work.—In the early years of the treatment, in accordance with the practice at Nordrach, walking was the only serious exercise permitted at most sanatoria. It was thought that in this way the needful exercise was supplied with less strain upon the chest and lungs than would be the case with muscular exercises involving the arms and their attachments to the chest wall. At Nordrach walking was extended in suitable cases to some sixteen miles or more a day, and, with the hilly surroundings and the graduated walks thus obtained, the results were very satisfactory. Walking is, however, apt to become monotonous, unless great variety of country is at hand; and experience has now shown that other forms of muscular exercise may be employed instead, without danger to the patient, and in many cases, indeed, to his immense benefit, both moral and physical.

At the Brompton Hospital Sanatorium at Frimley for patients of the poorer classes, Dr. Marcus Paterson, the

Medical Superintendent, devoted great attention to this subject, and devised and, with the approval of the Visiting Staff, put into practice a system of graduated labour, suited to the needs of the various patients. The labour is commenced as soon as it is seen that a patient can walk six miles a day without ill effects. The grades of work and exercise may be summarised as follows:

(1) Walking from half a mile to six miles daily; (2) carrying baskets of mould or other material, picking up wood, watering plants, etc.; (3) using a small shovel, cutting grass borders, hoeing, etc.; (4) using a large shovel, digging broken ground, mowing grass, etc.; (5) using a pickaxe, trenching, mixing concrete, felling trees, etc.; and (6) similar work to that prescribed in Grade 5, but for six hours daily instead of four as in the case of the preceding grades. In addition to the tasks thus allotted to them, the patients make their own beds, change their bed-linen, clean their wards and windows, polish the corridors, keep the dining-halls clean and the brass-work bright, and wash their plates, knives and forks after meals.[3]

The following account gives, in Dr. Paterson's words,[4] a more exact description of the amount of work done in certain of the grades:

"*Grade* 2.—Basket-work is subdivided into three sections. In the first the patient carries a load of about 12 pounds in weight a distance of 50 yards up a gradient of 1 in 10·7—*i.e.*, rising 14 feet in that distance. Such patients carry in a day eighty loads, or, in other words, they will carry 8½ hundredweight a distance of 50 yards. . . . In the second section the weight carried is about 18 pounds, the conditions being the same, and these patients carry about 13 hundredweight per day. In the third section the weight carried is 24 pounds. A patient on this work carries during the day about 17½ hundredweight for the same distance.

"*Grade* 3.—The small shovel is the ordinary coal scoop provided with a long handle. Patients commencing on this grade of labour will dig 2 tons of earth a day, and raise it 7 feet into a cart, and as they increase in strength will in a day lift about 4 tons the same height.

"*Grade* 4.—The large shovel is the ordinary shovel used by a navvy. Patients on this grade will dig and lift about 6 tons a day a distance of 7 feet.

"*Grade* 5.—Pickaxe-work is the hardest work possible, and consists of breaking unbroken ground, excavating, etc. Concrete-mixing comes under the heading of pickaxe-work, as, although the large shovel is used, the work is heavier than moving sand or mould.

"When a patient has been on a grade of labour for about three weeks, his fitness for harder work is considered. If the temperature has been normal, the weight satisfactory, the appetite good, and if he is feeling well (this to be determined by watching the way in which he performs his work), then he is put on harder work."

On these lines, the patients have carried out much useful work at the sanatorium, although many of them had before been engaged in clerical and sedentary occupations and had not been accustomed to manual labour. A reservoir capable of holding 500,000 gallons has been excavated and concreted. Trees have been cut down and sawn into firewood, the sanatorium has been painted, a kitchen-garden trenched and cultivated, and the grounds kept in order, each class of work being correlated to a certain grade of labour in accordance with the extent of muscular effort involved.

The results of this method of treatment have been gratifying. A large proportion of the patients, many of them the subjects of extensive but quiescent pulmonary disease, have been able to pass through all the grades of labour, and to do six hours of the hardest navvy work daily before leaving, during the last three weeks foregoing the hour's rest before dinner and supper, so that on returning home they are in a position to recommence work at once. As the labour is increased the patient's general condition improves, his cough and sputum diminish, and may eventually disappear. Especially we would emphasise the fact that in our experience the method, under the supervision of a careful Medical Superintendent, is free from risk. Hæmoptysis, contrary to what might have been expected, is not common, and from our experience at the Daneswood Sanatorium, we are inclined to think that it is less frequent than before the introduction of this form of treatment, which is also in use, though in a somewhat modified form, at this institution.

It sometimes happens, indeed, that a patient, when moved to a given grade, may find the work at first too much, or in his

eagerness to get well, may do more than he ought. Such conditions reveal themselves by headache and by a slight rise of temperature (a mouth temperature of 99·0° in men or 99·6° in women is regarded by Dr. Paterson as the "danger signal"), and if not checked would no doubt lead to extension of the lung disease. If the patient, however, is put to bed at once, kept at "absolute" rest, and treated in this respect as completely as if he were suffering from typhoid fever, after three or four days the temperature will again reach normal, and after the lapse of a few more days he may be able to return without ill effect to that grade of labour which before proved excessive. Such events emphasise the importance of the graduation of the labour and of the constant supervision of a skilled medical officer. In other and less successful cases it may not be possible, even after several attempts, for the patient to proceed beyond a certain low grade of labour.

In addition to the medical value of the treatment, we should like to bear testimony also to its immense moral effect upon the patients. The earlier sanatorium régime, especially when much rest and little exercise was practised, was not calculated, it must be admitted, to make them anxious to return to work, even though physically fit when discharged from the institution. Discontent in the sanatorium itself, owing to lack of occupation, was also by no means infrequent. Under a system of graduated labour, when its object has been fully explained, all is changed. The patients become cheerful, and take an interest in the scheme, which is at once economically productive and an agent in their recovery, and when they leave the sanatorium they are willing and in many cases able to recommence work at once. For these reasons the method has now been generally adopted in this country in sanatoria for the working classes, and with some modifications has also been introduced into certain private sanatoria.

We may now consider how the good effects resulting from a scheme of graduated labour are produced. It might be imagined that the result was simply due to the better general nutrition of the patient following upon a proper and due degree of exercise. This no doubt plays its part, but the observations of Dr. Inman, the Superintendent of the Laboratories of the Brompton Hospital, indicate that this is by no

means all, and that we are really dealing with a carefully graded *auto-inoculation* of tuberculin in gradually increasing doses, and with, in favourable cases, an immunising response on the part of the patient. Dr. Inman made numerous examinations of the blood in the cases under treatment, and he has shown that while early cases of tuberculosis with signs of activity exhibit marked variations in the opsonic index as the result of exercise (see p. 585), similar to those produced by a dose of tuberculin, those who had passed through the highest grade of work, and had lost cough and sputum, and whose disease was clinically arrested, showed no such sensitiveness of the index, but yielded an opsonic chart exactly resembling that of ordinary healthy individuals. He was able also in certain individual cases to trace this change in the index from abnormal to normal by examining the blood at different stages of the treatment, the index becoming normal when the disease is arrested, and when exercise no longer leads, as he believes, to absorption of tuberculin. His observations are of great interest, and support his contention that we are really dealing with auto-inoculation of tuberculin, and subsequent immunisation. This view is, moreover, sustained by the fact that, as he has demonstrated, exertion in these cases produces a response to the tuberculous opsonic index only, that relating to the staphylococcus showing no variation.

Diet.—We may now pass to the question of the diet which should be given in cases of phthisis suitable for sanatorium treatment. Not so very long ago it was the fashion, especially in certain German sanatoria, greatly to overfeed such patients, and at Grabowsee[5] the calorie value of the proteids, fats, and carbohydrates in the diet amounted to 1,200, 2,200, and 2,100 respectively, giving a total calorie value of 5,500. At Falkenstein[5], on the contrary, the similar figures were 530, 1,120, and 1,050, with a total value of 2,700.

Overfeeding is in contravention of a principle already laid down: that the body-weight and blood-volume cannot for long transgress the respiratory capacity without a corrective disturbance often difficult to control. Some few years ago Dr. Bardswell and Dr. Chapman, in conjunction with Professor Goodbody,[6] made some important observations at the Brompton Hospital upon metabolism in phthisis, and especially in

connection with the overfeeding which the disciples of Dr. Walther of Nordrach were at the time zealously upholding. Their results showed that "very large diets gave unsatisfactory results, as, although weight was gained, it was only at the expense of the general health, indicated by failure of appetite, more marked digestive disturbances, increased intestinal putrefaction, and in one case vomiting." Further, "this gain of body-weight was not associated with any more satisfactory progress in the tubercular lesion than was obtained with the smaller diets"[7]—an opinion which will be fully confirmed by physicians who have examined patients returned from sanatoria at which the system was practised, perhaps two stone above weight, but so breathless as to be scarcely able to cross the room. As a result of such observations, excessive overfeeding has now been largely given up.

In choosing a diet for a sanatorium patient, who is, as a rule, somewhat below weight, we shall not go far wrong if, as recommended by Dr. Bardswell and Dr. Chapman, who have devoted considerable attention to the subject, we choose a diet suitable for an average person in good health, but increase the proteids therein by 30 per cent., and the total energy-value by a similar amount. The extra calories required over and above that supplied by the increased proteid may be made up by the addition of either carbohydrates or fats—preferably, perhaps, the latter, when we remember how easily even very large quantities of fat are absorbed by phthisical patients. On these lines the diets at the King Edward VII. Sanatorium were constructed by Dr. Bardswell, then Medical Superintendent, and the results obtained have proved very satisfactory. The diets possess approximately the following nutritive values:

	Proteids (in grammes).	Fats (in grammes).	Carbo-hydrates (in grammes).	Calories.
Men	144	160	270	3,186
Women	126	160	220	2,814

In detail the diets are as follows:[8]

STANDARD DIETS IN USE AT THE KING EDWARD VII. SANATORIUM.

Time.	Men.		Women.	
	Diet.	Quantity.	Diet.	Quantity.
7.30 a.m.	Milk	½ pint	Milk	½ pint.
8.30 a.m.	*Breakfast.*		*Breakfast.*	
	Porridge (with milk)	½ pint	Porridge (with milk)	½ pint.
	Egg	1 (4 days a week)	Egg	1 (4 days a week).
	Meat (A), etc. ...	2 oz.	Meat (A), etc. ...	1½ oz.
	Bread	2 oz.	Bread	1½ oz.
	Butter	½ oz.	Butter	½ oz.
	Tea, coffee, marmalade, etc. ...	*q.s.*	Tea, coffee, marmalade, jam, etc.	*q.s.*
1.15 p.m.	*Luncheon.*		*Luncheon.*	
	Meat (B)	3 oz.	Meat (B)	2½ oz.
	Pudding (suet or milk)	5 oz.	Pudding (suet or milk)	3 oz.
	Bread	2 oz.	Bread	1½ oz.
	Butter	½ oz.	Butter	½ oz.
	Potatoes and vegetables, or salad	*q.s.*	Potatoes and vegetables, or salad	*q.s.*
	Stewed fruit, jam, etc.	*q.s.*	Stewed fruit, jam, etc.	*q.s.*
	Cheese and biscuits	*q.s.*	Cheese and biscuits	*q.s.*
4.30 p.m.	*Tea* (optional).		*Tea* (optional).	
	Tea, bread and butter, sandwiches, or cake	*q.s.*	Tea, bread and butter, sandwiches, or cake	*q.s.*
7.15 p.m.	*Dinner.*		*Dinner.*	
	Soup or fish (optional)		Soup or fish (optional)	
	Meat (C)	3 oz.	Meat (C)	2½ oz.
	Pudding (milk or suet)	5 oz.	Pudding (milk or suet)	3 oz.
	Bread	2 oz.	Bread	1½ oz.
	Butter	½ oz.	Butter	½ oz.
	Milk	½ pint	Milk	½ pint.
	Potatoes and vegetables	*q.s.*	Potatoes and vegetables	*q.s.*
	Stewed fruit, jam, etc.	*q.s.*	Stewed fruit, jam, etc.	*q.s.*
	Cheese and biscuits	*q.s.*	Cheese and biscuits	*q.s.*
9.30 p.m.	Milk	½ pint	Milk	½ pint.

N.B.—The weights given are the minimum quantities which are prescribed; second helpings of meat, pudding, and butter are allowed, if asked for. We may add that during and since the war margarine has been substituted for butter.

Meat (A). At breakfast, on different days of the week, one of the following is provided:

Bacon, ham, fish, tongue, or sausage.

Meat (B). At lunch this consists of one of the following:

Roast or boiled beef, hot or cold.

Roast or boiled mutton, hot or cold.

Beefsteak and kidney pudding, stewed steak or Irish stew, or liver and bacon.

Chicken, roast lamb, veal, or pork occasionally.

Meat (C). At dinner this consists of one of the following:

Hot roast or boiled beef, hot roast or boiled mutton, hot roast lamb.

When the sanatorium was first opened in 1906 a further half-pint of milk was given with luncheon, but after some experience this was discontinued, it being found that patients taking this quantity of milk tended to become too fat. The proper calorie value of the diet was maintained by increasing the helping of porridge at breakfast and the suet intake, and by the addition of extra vegetable proteid in the form of peas, beans, or lentils. On the dietary so modified it is found that patients below their normal weight usually gain about 1½ to 2 pounds a week. When some 6 pounds above their average normal weight, the diet is somewhat diminished, the amount of milk being still further reduced.

For the industrial classes, accustomed to fewer but larger meals, it is found at the Brompton Hospital Sanatorium that it is better to give the requisite nourishment in three solid meals—breakfast (8.15 a.m.), dinner (1 p.m.), and supper (6.30 p.m.), with a cup of tea at 4.30—rather than in smaller quantities at more frequent intervals.

Should the temperature rise during the treatment, and the patient be confined to bed, the diet need not necessarily be altered; but if the appetite fail, it may be tempted by varying the food. Thus, fish, chicken, sweetbread or mince may be ordered instead of the more solid joints, whilst the milk and suet puddings are replaced by junkets, custards, and jellies.

Alcohol is but rarely required by patients undergoing sanatorium treatment, but should the appetite flag, or the case "hang fire," a glass of Burgundy, port wine, or good beer at lunch and dinner may sometimes be prescribed with advantage. It is in these cases that the rest-hour preceding lunch and

dinner becomes of especial value, and it is a good rule with such patients to insist on its continued observance for some considerable time after returning home.

Medicinal Treatment.—The Dispensary at a sanatorium does not usually play a very important rôle. The early cases with but little active disease, which are best suited for the treatment, improve, as a rule, rapidly without medicine, though from time to time a mixture for dyspepsia, or more commonly a simple laxative, may be required. In the smaller percentage of active cases, which are to be found in all such institutions, drugs are of greater value, and their administration will be considered in a later chapter (p. 671). The question of combining tuberculin with sanatorium treatment will also be discussed (p. 711).

After a stay at a sanatorium for a certain time there are many people who may with advantage continue to live a country life, pursuing similar, but less rigid, lines of treatment. For such mitigated and, so to say, "peripatetic" cases of phthisis, which are often to be found at health resorts, it is our custom to lay down some such rules as the following:

They should take one hour's rest after breakfast, one before and one after lunch, and one before dinner. Their diet should be generous, and, if necessary, before the morning walk we would prescribe an ounce or two of raw-meat juice.* The afternoon tea may be directed to be made with milk instead of water, and at night, and perhaps also in the early morning, some malted milk food, such as Horlick's or Mellin's, may often be taken with advantage. Due exercise, regulated by the patient's strength, must be taken, and the open-air plan of treatment continued. On lines such as these consumptives belonging to the upper classes not uncommonly maintain their health, even though the pulmonary disease may be advanced.

REFERENCES.

[1] *An Essay on the Treatment and Cure of Pulmonary Consumption,* by George Bodington, surgeon. London, 1840. Reprinted by the New Sydenham Society, 1901, vol. clxxiii.

* This may be best prepared by squeezing the juice from half a pound of mutton or beef previously just browned at the fire to give the taste of cooking; it may then be served in a coloured claret glass, with one or two teaspoonfuls of port wine to flavour, and taken with a biscuit or finger of sponge cake.

[2] *On the Nature, Treatment, and Prevention of Pulmonary Consumption, and incidentally of Scrofula, with a Demonstration of the Cause of the Disease,* by Henry McCormac, M.D. London, 1855.

[3] The description is taken from—

(1) *The Sixty-eighth Annual Report of the Hospital for Consumption, Brompton,* 1909, p. ix.

(2) "Graduated Labour in Pulmonary Tuberculosis," by M. S. Paterson, M.B., *Transactions of the Sixth International Congress on Tuberculosis* (*Washington*). Philadelphia, 1908, vol. i., part ii., p. 886.

[4] "Graduated Labour in Pulmonary Tuberculosis," by M. S. Paterson, M.B., *Transactions of the Sixth International Congress on Tuberculosis* (*Washington*). Philadelphia, 1908, vol. i., part ii., p. 890.

[5] "Diet in Tuberculosis," by Professor Irving Fisher, New Haven, Conn., *Transactions of the Sixth International Congress on Tuberculosis* (*Washington*). Philadelphia, 1908, vol. i., part ii., p. 694.

[6] "On Metabolism in Phthisis," by Francis W. Goodbody, Noel D. Bardswell, M.D., and J. E. Chapman, L.R.C.P., *Transactions of the Royal Medical and Chirurgical Society,* 1901, vol. lxxxiv., p. 35.

[7] *Diets in Tuberculosis,* by Noel Dean Bardswell, M.D., and John Ellis Chapman, M.R.C.S., L.R.C.P., p. 37. London, 1908.

[8] (1) *Diets in Tuberculosis,* by Noel Dean Bardswell, M.D., and John Ellis Chapman, M.R.C.S., L.R.C.P., p. 59. London, 1908. See also

(2) *First Annual Report of the King Edward VII. Sanatorium,* 1906-1907.

CHAPTER XLIV

TREATMENT OF PULMONARY TUBERCULOSIS IN ITS EARLY STAGES—(*Continued*)

Results of Sanatorium Treatment.

We have sketched in broad outline the salient points in sanatorium treatment, and we have pointed out that for the great bulk of the population this treatment cannot be efficiently carried out except in sanatoria, at all events for the first few months; after which time the experience gained in personal management may enable patients of sufficient self-control and resources to continue the treatment at their own homes or in other convenient places. We cannot too strongly emphasise the educational value of sanatoria in inculcating hygienic methods of life, not only upon the patient himself, but upon all with whom he is brought in contact.

Let us now ask what are the results which have been obtained by this means, and how far it may be regarded as an advance on methods previously in vogue; and first of all let us consider the question in its relation to the industrial classes, in whom the conditions of life after leaving the sanatorium are often far from satisfactory.

Results of Sanatorium Treatment Among the Industrial Classes.—That treatment in a sanatorium is not, as was at first believed by some, a panacea for all forms of pulmonary tuberculosis, and in whatever stage of the disease, is now generally admitted. The experience of all who have to do with sanatoria, as well as the statistics from numerous institutions brought forward by Dr. Bulstrode[1] in his report upon this subject, unite in proving that it is only in the early cases that the most hopeful results can be expected. Advanced cases may derive temporary

benefit, and leave with gain in weight, and not a few with temporary restoration of working power. But after a few months, or it may be a year or two, the majority again break down, and succumb to their disease. This was well shown by Dr. John Gray in his "Summary of Results of the First Eight Years' Working of the Stanhope Sanatorium, Weardale, Durham"[2]—an institution opened in 1900 for the treatment of consumptive patients belonging to the industrial classes. He gives a table showing that, of 317 patients admitted during the years 1900-1908 with advanced disease (Stage III., Turban), not more than 130 were enabled to return to work after leaving the institution, and these, in most cases, followed it for a few months only. Within four years the bulk of the patients were dead. Occasionally such patients live longer, and may even surprise the physician by their vitality; but such an event is not of frequent occurrence.

In another table Dr. Gray shows that, of 267 patients admitted to the same sanatorium with early pulmonary tuberculosis (Stage I., Turban) during the same period, 239 were enabled to return to work after their discharge, and that in 1908 only 46 of the 267 patients admitted during the preceding eight years were known to be dead, 136, or rather more than 50 per cent., being still at work.

These conclusions have been universally confirmed, and it is in fact obvious that advanced cases of phthisis have already run their course half-way, or it may be two-thirds, towards the end before they come under the sanatorium régime, and that the fullest advantage of this régime can only be looked for in those who are admitted to treatment at the earliest period. Every effort must accordingly be made to persuade those suffering from phthisis to apply for treatment when still in the early stage of their disease, since the best results, whether immediate or enduring, are thus most likely to be secured.

We have dealt with this question in much greater detail in our last edition, and may here briefly say that the *general result of treatment in industrial sanatoria is that from* 50 *to* 60 *per cent. of the cases taken in the early stage are capable of working from four to five years after leaving the sanatorium* (see references [3], [4], [5] and [6]), the margin of difference chiefly depending upon the conditions of life to which they return.

On the other hand, most of the advanced cases would be dead within four years.[2]

The Importance of Efficient After-Care.—The value to be derived from sanatorium treatment, whether among the industrial classes or the well-to-do, depends in fact upon the efficiency of the "after-care." If the patient returns to the unsatisfactory conditions, whether in his work or at home, under which he acquired the disease, the improvement or arrest obtained at the sanatorium will infallibly be of short duration. This is shown in the better results obtained by Dr. Burton-Fanning[3] at the Kelling Sanatorium, Norfolk, where special care was taken from the first to obtain suitable employment for the patients after their discharge, and also by the similar results obtained among the employés of the Prussian and Hessian Railway Companies[6] quoted in our last edition.

The beneficial effect of *improving the work and environment* of the patient has also been recently demonstrated at the Brompton Hospital Sanatorium at Frimley. For this purpose the late Dr. Meek,[7] whose untimely death is much to be deplored, contrasted the after-histories of two groups of patients, in whose sputum tubercle bacilli had been found, and who were discharged from the sanatorium in 1915. In the first group, 32 in number, the patients were discharged with apparent arrest of the disease, and returned to the homes and working conditions under which they had previously broken down. Two years and a few months later 15 (46·9 per cent.) were still at work, 8 (25 per cent.) were alive but unable to work, and 9 (28·1 per cent.) were dead. In the second group of cases the disease was somewhat more extensive, and in 23 tubercle bacilli were still present on discharge, but the patients, 44 in number, left the sanatorium capable of doing a reasonably full day's work, and having a fair chance of maintaining working-capacity in healthy surroundings. Of these 44 patients, 29 effected a change of both residence and occupation, 5 a change of occupation only, and 10 found situations at their former employment, but in more suitable localities. In most instances the new work and surroundings were far from ideal, but constituted a marked improvement on their former conditions. After a few months over two years, 35 (81·4 per cent.) were well and at work; 6 (13·9 per cent.) were well and at work, but had been obliged

to have time off for illness; 1 (2·3 per cent.) was fairly well, but not at work; and 1 (2·3 per cent.) had died.

The figures quoted show the value of improving the work and environment of the patient on his return home. We have similarly been much impressed by the manner in which patients, whose health has been seriously undermined by extensive disease, but who have been taken on to the permanent staff of the sanatorium and have thus continued to live under the best conditions, are able for long periods to retain their health and working capacity. Several such patients are on the staff of the Frimley Sanatorium, and some have worked there continuously for eleven years. In another instance a skilled engineer was under the care of one of us at the Frimley Sanatorium during the year 1908, having extensive disease of the right lung; the following year he was appointed head engineer at one of our best-known sanatoria, and has remained in good health, working there continuously ever since.

It is the object of "colonies," such as the Cambridgeshire Tuberculosis Colony at Papworth Hall, under the supervision of Dr. Varrier Jones, to provide the patients who go to live there with suitable work under the best hygienic conditions, and we must wish success to this and similar institutions which recognise the fundamental importance of efficient after-care in the treatment of phthisis.

In country districts the inexpensive shelters of wood and canvas designed by Dr. Lyster,[8] of Great Baddow, near Chelmsford, which can be erected in the cottage garden, and in which patients can sleep, are helpful in promoting the continuance of the after-care.

Hints in Regard to Change of Work and Environment.—In advising upon the question of any change in work and environment after the patient leaves the sanatorium, care must be taken in the first place to see that the new work will be sufficient, with any other resources available, to enable the patient to obtain a living wage, and thus the full nourishment which his case requires. For this reason, as we have pointed out elsewhere,* it is generally a mistake to advise the patient to give up his former occupation and to undertake,

* The following paragraphs are quoted from an address delivered by one of us before the Inter-Allied Conference on the After-Care of Disabled Men,[9] 1918.

without any previous experience or training, work upon the land. The wages are not high, the housing accommodation is too often poor, and the work is at times exceedingly arduous. It is carried on also under conditions of exposure, and the hours at certain seasons are very long. As a result, only the exceptional case can stand it without breaking down, and it is not suitable for the average consumptive on leaving the sanatorium. We may add that it is not a life which possesses great attractions for the former town-dweller.

In many cases, owing to the difficulty of finding a new occupation, and the fact that under such circumstances the man will at first be untrained, and therefore capable, for a time at least, of earning only a low wage, the wisest course is to allow the patient to return to his former avocation, provided it be not clearly and obviously unsuitable to a consumptive patient, *changing, however, the environment whenever possible.* Thus, a clerk may return to his clerical work, but should obtain, if possible, a post in the country or at the seaside. Similarly, a bank clerk may often be transferred to a country or seaside branch. A railway porter should apply for removal from a city to a country station, and a policeman to a suburban division, while a carpenter may obtain work as an "estate carpenter."

Should it be necessary, owing to the special circumstances of the case, to find a fresh occupation for a patient, then one must be chosen in which the work is carried on under good conditions, and as much in the open air as possible. Whenever possible, too, the employé should be to some extent his own master in regard to the number of hours worked each day, and the ability to take a day off from time to time, should he not be feeling quite up to the mark. In this way his strength is conserved, and the danger of a breakdown lessened.

In deciding on the exact occupation, the various trades should be considered. For example, in the building trade the work of a painter or decorator, a builder, a bricklayer, a carpenter or joiner may all be permitted, by preference, however, in a smaller rather than a larger town. Wood-carving or wood-road laying may similarly be sanctioned.

In the transport trades a man might take up the work of a coachman, cab-driver, chauffeur, taxi-cab driver, motor-van

driver or motor-cleaner, but experience shows that the work of an omnibus- or tram-conductor or driver is generally too arduous in these days of large vehicles and rapid driving. Railway-ticket-collecting and certain forms of mechanical work on the railways are also suitable.

Although the work of an agricultural labourer is not, as we have seen, to be advised, certain lighter forms of work on the land may be recommended, such as market and flower-gardening, fruit-growing, hurdle-making, forestry and woodman's work. The occupation of a game-keeper, park-ranger, park-attendant or lodge-keeper is also excellent, but the number of such posts is limited.

Other light occupations which may be mentioned are those of a traveller, an insurance or commission-agent, rent-collector, canvasser (though in individual cases they may involve too many hours' walking), commissionaire and bookstall-attendant. Window-cleaning is not to be recommended, owing to possible risk of hæmorrhage.

The above list of occupations is by no means exhaustive, and is given only as an indication of the kind of occupation which may be recommended. For married men, we have often suggested that, if the wife is capable and has some knowledge of country life, some business, in which she can assist, may be entered upon in a country or village district.

We have drawn attention to the fact that relapse is only too common if the patient returns to the depressing conditions under which he acquired the disease. The converse is also true, and Dr. Picken has drawn attention to the fact that not a few soldiers who first showed evidence of phthisis in the army, and who had tubercle bacilli in the sputum, are still working regularly a year or two after discharge, and without having had the benefit of sanatorium treatment. In many the physical signs would now appear to be very slight. This result, as Dr. Picken[10] suggests, may well be attributable to early recognition and acceptance of the disease, and also to the fact that many of the patients possessed a fairly strong natural immunity to tuberculosis, and only contracted the disease under abnormal war conditions of mental and physical strain and exposure to infection. When removed from these conditions, the natural recuperative powers effect a cure.

The Value of Sanatorium Treatment among the Indus-

trial Classes.—It is pertinent now to ask how the results of sanatorium treatment among the working classes compare with those met with in patients of a similar class who have not had the advantage of institutional treatment.

Such a comparison is by no means easy to make, since adequate data are not available. Moreover, with improved medical treatment and the generally increased well-being of the industrial classes, coincident with the rise in wages which took place in the latter half of the nineteenth century, the prognosis of consumption has improved materially since Laennec and Louis a century ago placed the duration of the disease at about two years. Even in 1849, when the first medical report of the Brompton Hospital was published, the outlook was not much better than in Laennec's time, for of 215 cases which had terminated fatally in the hospital, the duration of 168 (78·1 per cent.) was under two and a half years, and only 6·5 per cent. of the patients lived longer than four years. Sixteen years later the researches of our late colleague, Dr. Pollock,[11] showed that the outlook was more favourable. Of 3,566 phthisical patients observed by him, *only* 129 (3·6 *per cent.*) *had died within two and a half years;* in the remainder the condition of the patients was still on the whole "favourable to the expectation of life for a considerable term."

In 1903 Dr. Stadler,[12] of Marburg, from his observation of 670 male and female patients during the years 1893-1901, was led to place the mean duration of life in cases of phthisis occurring among the working-class population of Marburg and its neighbourhood at between six and seven years.

Messrs. Elderton and Perry,[13] working under the auspices of Professor Karl Pearson, published in 1910 a statistical memoir dealing with this subject, in which they compared the results from sanatorium treatment with those obtained in pre-sanatorium days, using for the latter the data collected by the late Dr. Pollock and the late Dr. Theodore Williams. It should be noted, however, that the patients of Dr. Williams were private cases, and did not belong to the working classes. The conclusion arrived at was that "the mortality among sanatorium patients does not show any improvement over that of earlier days." The authors admit, however, that the comparisons are difficult by reason of the way in which the

older figures were given. In a more recent publication dealing with the private patients of the late Dr. Austin Flint in the United States between 1845 and 1870, Messrs. Elderton and Perry[14] draw attention to the fact that for reasons given any comparison between sanatorium results and presanatorium records is open to considerable criticism, and conclude that, while hoping and thinking that some improvement has taken place, it is far less than has sometimes been stated.

Our own experience would lead us to the conclusion that for *incipient and early stage cases* amongst the industrial classes superior immediate results are obtained in sanatoria, and that those results may be maintained for many years *under favourable conditions of working life.* The securing of these conditions constitutes the grave problem to be faced, and to this subject we have referred above.

Sanatoria for the industrial classes are not adapted for patients with advanced disease, nor for cases of pulmonary tuberculosis in an acute stage, nor for those more active cases, numbering, perhaps, some 10 per cent. of the whole, which steadily progress in spite of every form of treatment. Such patients are better in a hospital, and we believe that the best results can only be obtained when, as at Brompton, the sanatorium and hospital are linked together. In the hospital the patients can be carefully observed and grouped, those with fever and active disease being treated and nursed at the parent institution, those in an early and more quiescent stage, in whom a few weeks' observation gives promise of arrest of the disease, being drafted to the sanatorium. For patients with advanced disease further accommodation is required, and some suitable modification of portions of our existing poor-houses and infirmaries might easily be effected to assist in meeting the requirements.

Results of Sanatorium Treatment among the Wealthier Classes.—In patients belonging to the wealthier classes, whose circumstances after leaving home are, as a rule, much more favourable for the maintenance of health, the outlook, as might be expected, is more hopeful. Dr. Noel Bardswell,[15] in 1910, published the results which he obtained at the Mundesley Sanatorium in regard to the 241 male and female patients treated by him at that institution during the five years 1901

to 1905. These patients were unselected, and were admitted consecutively. His figures demonstrate clearly that with the well-to-do, as with the poor, the best results can only be looked for if treatment is commenced early. If also we examine the *early* or, as they are called, *incipient cases*, following the American method of classification, and note the results on January 1 of the fifth year after treatment in those discharged during the period 1901 to 1904, we obtain results which are comparable with those which we have already quoted. We find in this way that of 54 patients belonging to this group, 48, or 88·8 per cent., were still alive and fit for work between four and five years after their discharge, as compared with the approximate figures 50 and 60 per cent. obtained at the Stanhope and Kelling Sanatoria.

The results obtained over a period of twenty-one years at Dr. Trudeau's "Adirondack Cottage Sanitarium" in New York State, an institution which in some respects resembles the King Edward VII. Sanatorium at Midhurst, though the patients are of a somewhat higher social standing, were analysed statistically by Dr. Lawrason Brown and the late Mr. E. G. Pope.[16] They refer to 2,261 patients admitted in all stages of the disease. The immediate results were as follows:

TABLE SHOWING THE CONDITION ON DISCHARGE OF 2,222 PATIENTS ADMITTED INTO THE ADIRONDACK COTTAGE SANITARIUM, NEW YORK STATE, U.S.A.

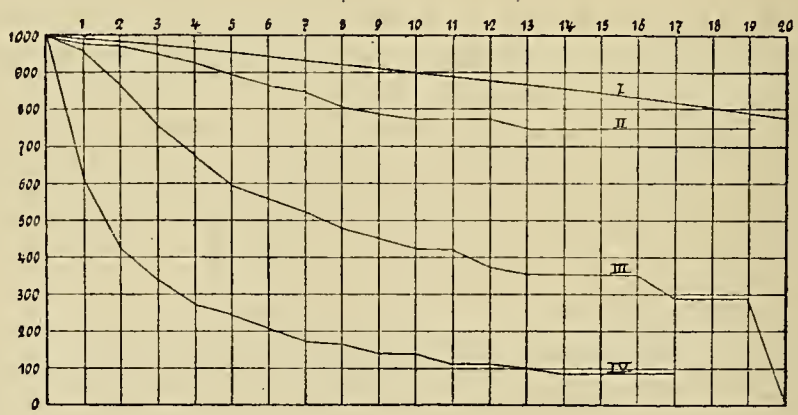
Condition on Admission.	Number of Patients.	Condition on Discharge.			
		Apparently Cured (per Cent.).	Disease Arrested (per Cent.).	Disease Active (per Cent.).	Died in the Sanitarium (per Cent.).
Incipient cases	620	56	32	11	0·03
Moderately advanced ...	1,329	12	46	40	2·00
Far advanced	273	—	16	78	6·00

The above figures demonstrate once again that for even the immediate results to be successful treatment must be commenced early. They also bring out the important fact, to which we have already referred, that *even in early cases arrest is not by any means a certainty.* Thus, of the 620 "incipient"

cases, 68, or 11 per cent., were discharged with the disease still showing signs of activity.

The further results, showing the after-histories of those who left the sanatorium (*a*) "apparently cured" (519 cases), (*b*) with "disease arrested" (890 cases), (*c*) with "active disease" (835 cases), may perhaps be best set forth in the following diagram, taken from Dr. Lawrason Brown and Mr. Pope's paper. The curves, based upon the Adirondack figures, show for 1,000 persons of each of the three groups, the numbers surviving at the end of successive years, the curves so obtained being compared with the expected death-rate of the general population.

DIAGRAM SHOWING THE NUMBERS SURVIVING AT THE END OF EACH YEAR, OUT OF (I.) 1,000 PERSONS IN THE GENERAL POPULATION; (II.) 1,000 DISCHARGED "APPARENTLY CURED" FROM THE ADIRONDACK COTTAGE SANITARIUM; (III.) 1,000 DISCHARGED "ARRESTED"; (IV.) 1,000 DISCHARGED WITH "ACTIVE DISEASE."



If we study the curves shown above, it will be seen that the patients who have been discharged "apparently cured" (Curve II.) possess an outlook of a very hopeful kind, the number surviving at the end of fifteen years being about 750 out of every 1,000, as opposed to 850 per 1,000 of the general population. A glance at the table on the preceding page will, moreover, show that of the 620 patients admitted in the *incipient stage of the disease*, whom careful diagnosis should at the present day at once bring under treatment, 56 per cent., or more than half, were discharged "apparently cured," and possess therefore this favourable prognosis.

Curve III. further reveals that the patients who were discharged with "arrested" disease, which group includes 32 per cent. of the incipient cases, still possessed an average expec-

tancy of life of between seven and eight years. In only 11 per cent. of early cases was the outlook gloomy, as shown by the rapid fall to the base line of Curve IV.

Such results are satisfactory, and demonstrate what may be hoped for from sanatorium treatment in the upper classes, whose members are able to carry on the treatment after leaving the institution, and in whom the disease has been detected and brought under treatment in an early stage. With the more advanced cases the future is proportionately less bright.

Data derived from the Records of the King Edward VII. Sanatorium, Midhurst.—A newer method of estimating the results of sanatorium treatment was followed by Dr. Noel Bardswell,[17] in collaboration with Mr. J. H. R. Thompson, a Fellow of the Institute of Actuaries, under the auspices of the Council of King Edward VII. Sanatorium, Midhurst, and carried on since 1914 with the countenance and aid of the Medical Research Committee of the National Health Insurance, who have borne the expense of publishing the results. The lines of the investigation are similar to those adopted by Messrs. Elderton and Perry[14] in their study of the results obtained at the Adirondack Sanitarium to which we have alluded. The cases are grouped "1," "2," "3" for each sex, according as they are in the "incipient," "moderately advanced," or "far advanced" stage. The cases include all those, 1,707 in number, which were discharged from the sanatorium, after not less than eight weeks' treatment, during the eight years 1907-1914. With such diligence has the inquiry into the after-history of the cases been prosecuted that in only 3·5 per cent. has it failed to be obtained. The important feature in the inquiry is the comparison made at each year of the mortality among the various groups of patients with that which would have been expected had the groups been samples of the general population corresponding as to age and sex. We must refer the reader to the full report, with its numerous elaborate tables, and need here only give the results. They are briefly as follows:

1. The mortality experienced by patients after sanatorium treatment is very heavy, the death-rate, even for patients admitted in the early stage (group 1), being nearly six times, that of patients admitted with moderately advanced disease

(group 2), over sixteen times, and that of patients admitted with far advanced disease (group 3) thirty-eight times greater than the rate for the population of England and Wales.

2. The results for male and female patients show no material difference, but the mortality is relatively greater at the younger ages at discharge.

3. The results at Midhurst are generally similar to, and are confirmed by, those obtained by Messrs. Elderton and Perry[14] from their actuarial investigation of the patients at the Adirondack Sanitarium in New York State to which we have already referred.

4. Another important conclusion is that the mortality is greatest during the first *two or three years, following discharge*, the excess mortality being subsequently materially reduced, so that for group 1 at least the rates for durations of six to eight years after discharge are not seriously above the normal. This is shown in the following diagram taken from Dr. Bardswell and Mr. Thompson's paper:

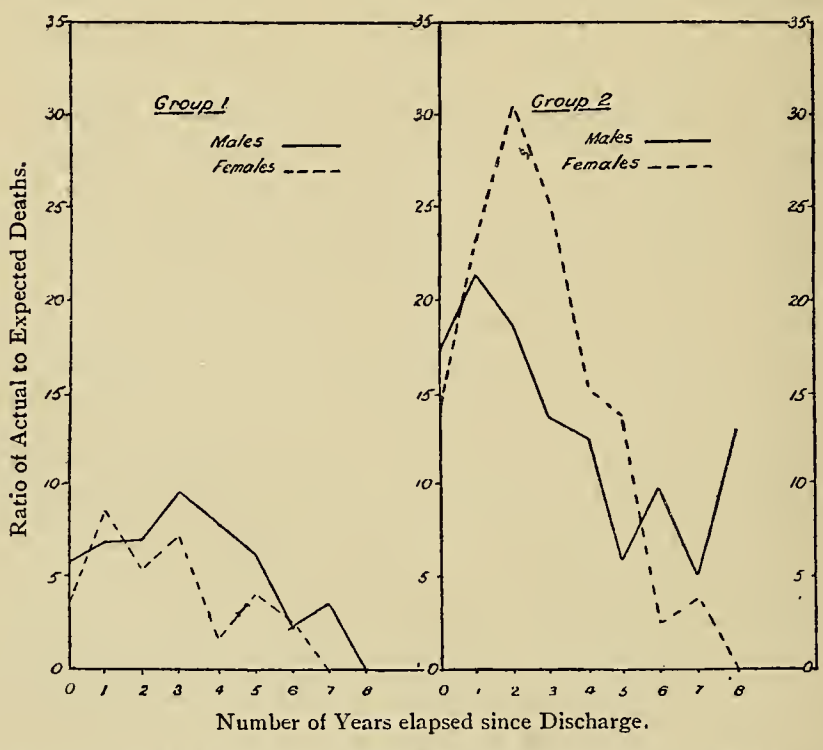


FIG. 64.—DIAGRAM SHOWING THE RATIO OF ACTUAL TO EXPECTED DEATHS, DETERMINED ACCORDING TO THE NUMBER OF YEARS WHICH HAVE ELAPSED SINCE DISCHARGE FROM THE SANATORIUM, AND INDICATING THAT THE MORTALITY IS GREATEST DURING THE FIRST THREE YEARS, AND THAT THIS PERIOD IS THEREFORE THE CRITICAL ONE FROM THE AFTER-HISTORY POINT OF VIEW.

It follows from the above that the first three years are the "*critical period*" for patients after discharge, and that during this time they must take no liberties with themselves, but do all in their power to continue to live on sanatorium lines.

5. Another valuable conclusion is that for patients discharged with the disease arrested, the subsequent mortality is not very different for groups 1, 2, and 3, showing that in such cases the extent of the disease on admission is of minor importance; but that on discharge, if the disease be not arrested, the condition at the commencement of treatment governs the prospect of longevity. Thus in making a prognosis the condition on discharge is at least of equal importance to that on admission.

6. In their report Dr. Bardswell and Mr. Thompson show that in two-thirds of the patients the disease commenced insidiously, and in one-fifth with hæmoptysis, and that in the latter patients the mortality experienced was lighter. This is in accordance with general experience, and is due in part at least to the early recognition of the disease which hæmoptysis entails, and the more prompt treatment which generally follows.

7. As might be expected, the records prove that patients with laryngeal as well as pulmonary tuberculosis experience substantially higher rates of mortality than cases not so complicated; but in the comparatively uncommon cases of early laryngeal tuberculosis, associated with but slight lung trouble, the outlook is not unfavourable.

8. The results further show that the prognosis is materially improved if tubercle bacilli disappear from the sputum before discharge. Whenever possible, therefore, treatment should be continued until a negative sputum has been obtained.

9. Lastly, it is interesting to note that, as Messrs. Elderton and Perry[14] also found in their study of the Adirondack patients, the prognosis in a given case is not made worse by a consumptive family history. Messrs. Elderton and Perry point out that this does not mean that the children of tuberculous parents are not more likely to acquire the disease than patients without such family history, but that if an individual possesses the type of constitution suitable for the development of tuberculosis, it is immaterial whether such constitution is traceable to the parents or due to disease or some other cause.

In conclusion, we would repeat that we have for clearness' sake spoken above of sanatorium treatment as representing treatment in a sanatorium. We have already, however, pointed out that a certain proportion of the well-to-do can secure the conditions of such treatment in their own homes, or at least can secure the after-care treatment on the same lines at an earlier period and for a longer time than can less favoured persons. Indeed, we would urge that sanatorium treatment is only the enforcement of proper hygienic measures under medical supervision.

It will be within the experience of physicians to have seen cases of pulmonary tuberculosis amongst both out-patients and the better classes who, without any formal sanatorium treatment, but simply acting under advice, have recovered from this malady. We are ourselves aware of many such cases of complete recovery, standing the test of twenty to thirty years of time, and it may be of interest to quote the two following, in each of whom, however, climatic change formed an element in the treatment:

Case I.—Mrs. X. consulted Dr. Douglas Powell in October, 1891, on account of some hæmoptysis. There had been preceding symptoms of only a few weeks' duration. The physical signs showed early lesion at one apex, but the sputum revealed the presence of tubercle bacilli in fair numbers. The lady went to St. Moritz in November, and remained there for the winter. She greatly improved, and completely lost her cough. She lived a careful life under Dr. Holland's observation at St. Moritz, but was under no "sanatorium" régime. She returned to St. Moritz in January of the following year for a couple of months as a measure of precaution, but not on account of any return of symptoms. She resumed her professional work. In 1894 she again came under observation with a severe attack of enteric fever with relapse, and has since then passed through other severe illnesses. She has never, however, had any return of chest symptoms, and is now (1920) in fairly good health, having recently accomplished a visit to America.

Case II.—An officer, aged 35, had a severe hæmoptysis in 1887, followed by cough and expectoration, with tubercle bacilli present in the sputum, and other symptoms of early tuberculosis. There were pyrexial signs, dulness and slight crepitation at the right apex. He was a man of athletic build, 6 feet 2 inches, although rather narrow-chested for his height. After a period of treatment he went by sailing-ship round the Cape to Australia and New Zealand, starting at the end of September, arriving in Melbourne

on December 22. He returned by steamer, spent a short time at Aix-les-Bains, and arrived home at the end of May, 1888. He did not gain much by the voyage, and spent the winter of 1888-89 at St. Moritz, with benefit, and the following winter on the Riviera, and after that went to India and round the world. There were no sanatoria available in those days, but he was a man of temperate habits and self-restraint, and led a careful sanitary life. By advice he relinquished the army on his return from abroad and went to live on his own estate in the North, and having all the attributes of a sportsman and a country gentleman, he spent his leisure time fishing, shooting, etc. He regained health, and in course of time he could out-walk his keepers grouse-shooting, shooting over dogs, and deer-stalking. Except for an occasional attack of influenza, his health has remained excellent.

In January, 1919, he had a bad attack of influenza with bronchitis, and on one occasion in the earlier days of this attack a specimen of sputum sent for examination by his medical adviser was reported to contain "a very few tubercle bacilli." On another examination within a week, however, the report was negative, and when seen by one of us at the end of February the report on a specimen of sputum was "no tubercle bacilli were seen in this mucous sputum after a prolonged search." He had completely recovered, and there were no physical signs beyond slight impairment of percussion note over the right apex. He was now 67 and leading the same active country life.

Here is a case of recovery from tuberculosis and acquired immunity of thirty-two years' duration, developed under healthy conditions of life and maintained under medical advice intelligently pursued, although not with the system and formality of sanatorium therapy. We have no doubt that the visit to the Engadine started the cure, as has been the case with others in our experience. Assuming the accuracy of the report as to the presence of a few bacilli on one occasion during the last influenzal attack, we do not think it a matter for surprise, although when examined on other occasions, and within a week and again in a month, no bacilli were found, for it is quite conceivable that a catarrhal attack affecting an old quiescent lesion might loosen from it some effete products containing bacilli. In any case, the immunity is shown in their failure to gain ground.

We have alluded to other cases treated on similar lines and enjoying many years of immunity, and we might mention not a few more, some with a very considerable degree of lesion, but they by no means invalidate our opinion that, taking numbers, and considering the conditions under which tuberculosis is most generally acquired and prevalent, sanatorium treatment is the best and most rational average treatment of the disease. We only note that many isolated cases have informally, and as it were by chance, by the adoption of some-

what similar lines, found immunity. One result of the institutional treatment of phthisis has been to educate the public and the profession in sanitary methods of self-management in this disease, and to it we owe much of the improvement of those who do not rigorously adopt the régime.

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CHAPTER XLV

ON CLIMATIC CHANGE IN THE TREATMENT OF PULMONARY TUBERCULOSIS

We must preface what we have to say in regard to the benefits to be derived from climatic changes with the warning that, owing to recent legislative enactments, the scope of this valuable method of treatment has been considerably curtailed. In the Dominions of Australia and New Zealand, in Canada and the United States, the immigration laws now forbid sufferers from tuberculosis to enter the country if the disease be in an infectious stage, and at the present time (May, 1920) the subjects of pulmonary tuberculosis, in whatever stages, are not allowed admission into the Union of South Africa. Precautions are also taken to insure that no patient shall become a charge upon public funds. In every case, therefore, we should advise the intending traveller or settler to take the precaution of communicating before he leaves England with the chief medical officer of the Commonwealth Medical Bureau at Australia House, Strand, or the corresponding authorities attached to the New Zealand, Canadian, United States and South African Emigration Offices in London, so as to assure himself that he will be permitted to land on arrival at his journey's end.

It should be borne in mind also that, although the ensuing pages are written more especially with reference to pulmonary tuberculosis, all other chest diseases in which change of climate is desirable are taken into account.

We have seen in the last chapter that in the majority of cases of early phthisis it is well to recommend, for a time at least, sanatorium treatment. By this means the patient is taught in a practical manner how best to conduct his life, and is safely guided through the earlier and often febrile period

of his disease, during which, without the minute and careful supervision which forms so essential a feature of the sanatorium régime, he would be liable, through imprudence or ignorance, to aggravate his trouble.

Until the more acute symptoms have completely passed away and the malady has become quiescent, a sanatorium in England, which does not involve a long journey, is to be preferred. When, however, after some months, this stage has passed, and when the temperature has become normal, and remains so even after a considerable degree of exercise, then climatic change, whether combined with further sanatorium treatment or not, may be considered.

The climatic resorts to which such patients may be sent differ with the time of year, the nature of the case, and the temperament of the patient. It is difficult at the present moment to classify these resorts with exactness; but there are certain indications for selection generally applicable, which we will endeavour to point out.

Elevated Climates (above 3,000 Feet).—The first question which often presents itself for decision is whether to send the patient to an elevated climate or not. It will clear the ground, therefore, if we first briefly consider these climates, and the cases that should and should not be sent to them.

Alpine Stations.—The most important of these are *Davos* (5,100 feet) and *Arosa* (5,900 feet) in the Canton Grisons; *St. Moritz* (6,000 feet) in the Upper Engadine; *Montana* (5,000 feet), above Sierre, in the Canton Valais; and *Leysin* (4,800 feet), not far from Aigle and the Lake of Geneva. At a lower level, and therefore with a somewhat milder climate, we may mention *Les Avants* (3,200 feet), *Caux* (3,500 feet), and *Château d'Œx* (3,260 feet), all situated in the Canton Vaud, not far from Montreux.

At Davos there are various sanatoria to which the patient may be sent, if thought desirable, including the one upon the Schatzalp, situated 1,000 feet above Davos itself; there are also good hotels fairly well adapted for pursuing sanatorium lines of treatment. At Arosa there are several sanatoria and good hotels. At Montana the Palace Hotel, originally built as a sanatorium, has been recently reconverted to its original use, and is now run on sanatorium lines; the climate is very dry, with abundant

sunshine, but the air is not so still as at Davos. St. Moritz is better adapted for increasing the resistance of persons not as yet actually attacked.[1]

It is during the winter months that the properties of these climates, which are regarded as remedial in phthisis, are especially manifested. These properties are: (1) low atmospheric pressure, some five inches of mercury below the barometric measurement at the sea-level; (2) dryness of atmosphere; (3) purity of air, as shown by freedom from organic and inorganic dust; (4) aseptic qualities by virtue of freedom from organic germs and the relatively large proportion of ozone present; (5) low temperature of the air; (6) increased transparency to the sun's rays, both illuminating and chemical (diaphaneity and diathermancy), from the thinness and clearness of the atmosphere and the diminished amount of aqueous vapour contained therein; (7) stillness of the air, in consequence of which its coldness is less felt. In the meteorological table on p. 643 certain of these factors are demonstrated.

The beneficial effects of these climates are attributable to the rarefaction of the air as a stimulant to the respiratory function; to its aseptic qualities, by virtue of which putrefactive and fermentative processes are hindered; and to the vivifying influence of the brilliant and warm sunshine, which enables patients to be much out of doors. One effect of the climate is seen in the blood-count, which normally at Davos registers six million red cells, with a percentage of hæmoglobin ranging to 105 per cent. or even higher.

The diaphaneity and diathermancy of the rarefied atmosphere of these high regions is an important fact, to which Dr. Denison and Sir Hermann Weber[2] were among the first to draw attention. By virtue of this quality, due especially to the smaller amount of aqueous vapour present in the atmosphere, the rays of the sun pass through with increased readiness, powerfully affecting objects exposed to them, although heating the air itself but little during their passage. Dr. Denison[3] put the matter shortly by giving the following rule of increasing diathermancy of air, viz.: "For each 1,000 feet rise in elevation there are about four degrees greater difference between the temperatures in the sun and in the shade on perfectly clear days at 2 p.m., as recorded by the black metallic-backed thermometers, other influences than those of

sun and shade being excluded." Sir James Dewar has shown the intensely destructive power of the ultra-violet rays upon morbific organisms, and these rays are in the higher altitudes more potent because less obstructed.

Improved sanguification and nutrition, expansion of unaffected parts of the lungs, and restriction of the limits of diseased areas, are results obtained in a striking degree in successful cases by residence in these localities. They are, however, rigorous climates, and it must not be forgotten that, potent for good in well-chosen cases, they are also active in working mischief to those whose condition is not well adapted for them. It cannot, moreover, now be claimed, as was once asserted, that these climates possess any exclusive power, by virtue of rarefaction of atmosphere, of developing healthy lung and contracting diseased areas. We have known patients recover under the most diverse climatic conditions, and have often observed the development of unaffected lung proceed to the utmost possible limits in patients who have never been at a higher level than the galleries of the Brompton Hospital. Such patients have found their own salvation in acquired immunity. The physical state common to all such cases is contraction of the limits of disease, and compensatory enlargement of the opposite and of the free portions of the same lung, and we doubt the possibility of exceeding the compensatory development that may be observed in these favourable cases of phthisis arrested in the plains. Nevertheless, the statistics of Dr. Theodore Williams,[4] the results of treatment in the Swiss sanatoria,[5] and our own experience would sanction the view that in suitable cases these good results may be best obtained by high-altitude treatment, and we are only now desirous of pointing out that rarity of atmosphere, although it may be an auxiliary, is by no means an essential condition for bringing about this much desired end. We have elsewhere emphasised the importance of distinguishing between merely enlarged or dilated lung and truly hypertrophied lung, in association with localised or arrested disease of some other portion. There is no advantage, but much the contrary, in producing emphysema in phthisis.

The cases suitable for treatment at elevated resorts are those of threatened disease and localised tuberculosis in the early and the quiescent stage, especially when associated with

a languid temperament and a moderate degree of anæmia. Cases of hereditary tendency, defective thoracic conformation and capacity, incomplete recovery from acute or subacute inflammatory affections of the chest, whether pleuritic or parenchymatous, are peculiarly adapted for such treatment. Cases of more advanced tuberculous disease, if it be restricted and inactive, are also suitable, provided that the patient have sufficient vitality to respond to the extra demand made upon his system by the rigorous climate. It is important that the reserve lung be sound, not emphysematous, and that the heart and vessels be healthy.

Cases which have commenced with hæmoptysis, and in the course of which hæmoptysis has from time to time occurred from active or passive congestions, are not thereby disqualified for residence in high climates, but a few weeks should elapse before removal there. For cases of recurrent hæmorrhage, however, in which an ectasia or aneurism of a pulmonary vessel in a localised cavity is suspected, such climates should not be suggested. The following conditions are also unsuitable for residence in high altitudes—viz., the acute stage of phthisis in any form; the erethic* constitution in any stage of the disease; cases in which the larynx is affected; patients with advanced phthisis; those in whom the tuberculous disease is complicated by marked emphysema, albuminuria, or intestinal ulceration, or associated with signs of general bronchial irritation; those in whom the malady makes its appearance in later life.

The winter season is the best to choose for residence in these localities, but in those cases in which benefit is experienced, continued residence through the summer at about the same elevation, and a second winter in the same locality, should be decidedly advised. A third or fourth year may sometimes be thus well spent. In cases of mere delicacy or threatened disease during convalescence from some acute chest malady, a single short period of residence may suffice. In early-stage cases, which have derived benefit from a single winter, the good result is often maintained by two or three months' return to the same locality for several succeeding winters. In this practical world but few people can afford

* Nervous, irritable subjects, neuralgic, dyspeptic, bad sleepers, with habitually quick pulse and dry, harsh skins.

to live indefinitely in idleness above the clouds! Persons who have recovered after a season or two at the Alpine stations may sometimes live and earn a living in the highlands of South Africa, to which we shall refer later.

Patients should as a rule proceed to their proposed winter-quarters on high ground not later than the end of October, so that they may avoid the dampness and fogs which at this season of the year are so characteristic of our English climate. They should not return home before the commencement of May, the winds in England during April often proving very trying. It is a not uncommon practice for patients to leave the high Alpine resorts in March, when the snow is melting, and to stay at some lower level, such as *Thusis*, *Promontogno*, *Ragatz*, or *Glion*, before returning home. But such a practice is not essential. It is most important to warn patients against wandering about in a restless fashion, visiting other European stations at the end of their treatment in high altitudes. Our experience is that such cases often suffer relapse, and return in much the same condition as that in which they left home.

Surgical Tuberculosis.—The benefits to be derived from a residence in Alpine stations are not confined to cases of pulmonary tuberculosis, and, as Rollier[5A] and others have shown, remarkable results are obtained at Leysin (4,800 feet) in surgical tuberculosis, including affections of the bones, joints, spine and glands, and also such manifestations as tuberculous peritonitis and disease of the genito-urinary tract. In such cases, in addition to the general climatic influence, direct treatment is carried out by gradually exposing the whole body, as well as the part affected, to the immediate action of the sun's rays for increasing periods of time, and to this form of treatment the name "*Heliotherapy*" has been applied.

The Rocky Mountains.—Among the elevated resorts in North America may be especially mentioned the Rocky Mountain stations, *Colorado Springs* and *Denver*, situated in Colorado State at an elevation of between 5,000 and 6,000 feet. Three miles from Colorado Springs, and facing Pike's Peak, the Cragmor Sanatorium has been built at an elevation of 6,100 feet. The climate of this region resembles in many respects that of the Swiss resorts, but, as may be seen by a study of the meteorological data (p. 643), it is several degrees

warmer in winter, the mean temperature for the six months at Denver being 36·5° F., as compared with 27·2° F. at Arosa, and 25·9° F. at Davos. Snow, consequently, is not often seen. A much greater amount of sunshine is also enjoyed, but against this must be set the fact that the region is decidedly more windy, and dust-storms, often of a peculiarly irritating character, are not infrequently met with.

Other stations in the Rocky Mountains are *Oracle* (4,500 feet) in Arizona, and *Santa Fè* (7,000 feet) and *Las Vegas* (6,400 feet) in New Mexico. *Manitou Park* (7,500 feet) and *Estes Park* (6,800 feet), in Colorado, are also favourite summer resorts.[6]

South American Andes.—At *Jauja, Tarma, Huancayo,* in the Peruvian Andes, at *Santa Fè de Bogota,* and at *Quito,* in Ecuador, not far from the equator, all at an elevation of about 10,000 feet, the effects of a highly rarefied air are obtained with a temperate, equable and brilliant climate.[7] Only in rare instances, however, would one suggest such elevations for invalids.

The South African Highlands.—These highlands possess climates of the greatest value for the sufferer from tuberculosis, though at the present time such cases are not welcomed in the Union. The benefits of the climate may, however, be enjoyed by those who have merely an inherited tendency to the affection, and such patients may here develope into robust health, the dry, warm summer and temperate winter climates, which these high upland regions possess, rendering them suitable for permanent residence, more especially for those who desire to escape our cold winters. Before recommending South Africa for prolonged residence, we should remember that the ordinary necessaries of life in that country are expensive, and that employment may not be easy to obtain. Unless, therefore, a patient possesses private means, or has friends in the country, or some definite work in view, we may well hesitate before suggesting a residence there, however suitable on other grounds the case may seem. It is, perhaps, hardly necessary to add that it is absolute folly for those in active or advanced consumption to think of venturing upon the fatigues and risks of so long a journey, and it would be exceedingly improper to advise them to do so, even were it probable that they would be permitted to land. Private sana-

TABLE GIVING THE METEOROLOGICAL DATA FOR VARIOUS HEALTH RESORTS, THE STATIONS BEING ARRANGED IN THE ORDER OF THE MEAN TEMPERATURE OF THE SIX WINTER MONTHS.

	Mean Temperature.						Averages during the Six Winter Months, November to April.					
							Temperature.		Relative Humidity.	Rainfall.		Sunshine.
	Nov.	Dec.	Jan.	Feb.	March.	April.	Mean.	Range.		Amount.	Days.	
	Degrees.	Degrees.	Degrees.	Degrees.	Degrees.	Degrees.	Degrees.	Degrees.	Per Cent.	Inches.		Hours.
Helwân ...	65·9	57·3	55·0	57·2	61·6	70·0	61·2	21·0	64	1·53	7	1,334
Funchal ...	63·3	60·2	58·6	58·1	58·7	60·2	59·9	12·7	65	21·34	53	—
Algiers ...	63·1	57·0	54·8	56·3	58·7	62·4	58·7	13·0	66	22·98	84	—
Los Angeles ...	60·0	56·0	54·0	55·0	57·0	60·0	57·1	21·2	71	14·20	32	1,457
Mentone ...	54·6	49·6	47·9	49·6	52·3	58·5	52·0	12·3	—	16·87	47	—
San Remo ...	54·4	49·7	46·6	48·9	51·1	56·9	51·3	—	—	—	—	—
Cannes ...	52·6	46·3	48·0	48·8	51·0	55·5	50·4	12·7	73	19·11	43	—
Nice	53·2	47·5	45·6	47·2	50·8	55·9	50·0	18·6	75	15·49	43	—
Torquay ...	47·9	44·6	42·5	42·7	44·5	48·4	45·1	10·0	82	17·37	90	568
Hastings ...	45·3	40·8	39·5	40·1	42·4	47·0	42·5	9·2	85	14·50	101	580
London ...	43·8	39·7	38·6	39·5	42·2	47·3	41·9	10·6	84	11·31	83	273*
Denver ...	39·0	33·0	29·0	32·0	39·0	48·0	36·5	25·5	59	5·10	35	1,327
Arosa	32·0	25·2	22·6	23·4	27·0	33·6	27·2	—	65	18·95	70	715
Davos	29·3	21·0	18·7	22·8	27·3	36·0	25·9	—	81	13·07	—	715

For further details in regard to the climatology of the above stations we must refer the reader to the meteorological tables printed in the Appendix to the last edition of this work. As the figures relate in nearly all cases to a considerable number of years, they may be regarded as reflecting with considerable accuracy the climatic features of the various resorts. A reservation must, however, be made in regard to the mean relative humidity; the observations upon which these figures depend are not taken at the same hour in every country, and are not, therefore, strictly comparable.

* Recorded at Westminster.

toria exist at *Modderfontein* near Johannesburg, and at *Belfast*, both in the Transvaal; at *Nottingham Road*, a healthy farming district in Natal, forty miles from Maritzburg; at *Laingsburg*, in the Cape Province, and no doubt in other places.

The South African resorts may be classified into those of the Cape, Natal, the Orange Free State, and the Transvaal Provinces. There are many localities, and will, no doubt, in the future be many more, available for the sojourn or settlement of those who cannot enjoy full health in their own country, yet who are not in any actual or active degree of pulmonary unsoundness. We can only here refer to a few places which are of ready access, and are suitably equipped for the accommodation of the degree and kind of invalidism which we have in mind.

The Province of the Cape of Good Hope.—Cape Town itself, seventeen days' voyage from Southampton, is entirely unsuited for chest invalids. As pointed out by Mr. Samler Brown,[8] in his interesting and valuable guide to South Africa, it is, owing to its western aspect, swept by the cold Antarctic current, and the climate is consequently cold, windy and treacherous, although the mean annual temperature is about the same as that of Naples. It will, of course, be remembered that the summer months of South Africa are from November to May, when the autumnal rains begin. *Simons Town*, on *Simons Bay*, is the first southern station we need mention, and although only twenty miles from Cape Town, it is washed by a sea current from the Indian Ocean from 10° to 12° warmer than that which bathes the western shores, including Table Bay. With a mean maximum annual temperature of 70·4° and minimum of 57·1°, and a rainfall of 29·3 inches, it forms a good resting-place, and also one in which certain delicate patients may reside throughout the year. South-eastern winds are somewhat of a drawback during the summer months, and during their prevalence protection can be sought by a change to Wynberg, a healthy suburb eight miles from Cape Town, which is protected from these winds by the shoulder of Table Mountain.

The highland districts of the Cape Province are, however, more desirable for chest invalids, and the Cape to Cairo Railway, which runs north-east to the western border of the Orange Free State and the Transvaal, skirts the great Karroo

tableland at an elevation of 3,000 to 4,000 feet, and along or within easy reach of its route are several places of varying altitude which may be recommended. The high-ground climates of South Africa, the stations of which we are about to consider, possess, like other elevated regions, a low atmospheric pressure, abundant sunshine with great diaphaneity and diathermancy of the atmosphere, and are further remarkable for dryness, the rain falling principally in storms, chiefly in the autumn (our spring). Owing to their geographical position, they are very much warmer than the Swiss mountain resorts; but they are also more dusty and windy.

Ceres (1,493 feet), eighty-four miles from the Cape, a small, well-laid-out town, with good water-supply, possesses a warm, dry summer climate (corresponding with our winter), with a mean shade temperature of 66·5°, a sandy soil, abundant sunlight, and cool nights, with but little mist; it is, moreover, sheltered from prevailing winds. The rainfall, 41 inches, occurs mainly in the winter, the mean temperature of which is 51·7°.

Beaufort West (2,850 feet), some 150 miles farther on, is also a well-found town, but with more arid and less picturesque surroundings.

Graaf Reinet (2,463 feet), more central in the Province, can be reached through the De Aar Junction, but is more accessible direct from Port Elizabeth, from which it is distant 185 miles. It is a well-found townlet of some 7,000 inhabitants, possessing a warm, dry, bright and sunny climate, with well-laid-out botanical gardens, sporting clubs, sheep and ostrich farms, home-irrigated land, forming "a veritable oasis in the dry Karroo Veld."[8] The mean summer temperature (November to April) is 69·7°, that of winter (May to October) 57°; the total rainfall 17·15 inches in fifty-six rainy days. *Somerset East* (2,400 feet) is another and smaller agricultural town, with similar features to Graaf Reinet, picturesque surroundings and excellent climate. *Middleburg* (4,095 feet), 250 miles from Port Elizabeth, similarly situated amid mountain and river, is an agricultural centre with a more stormy summer and a colder winter, possessing attractions for more robust persons.

Aliwal North (4,350 feet), in the north-east corner of the Province, situated on the Orange River, and bordering the

Orange Free State Province, is one of the choice spots for residence. It is best approached by rail from East London (282 miles), and is in direct communication with the Orange Province by a bridge across the river. Aliwal North is a fine well-equipped town, with churches, schools, and good recreation-grounds, in a well-watered, fertile neighbourhood, with outlying centres of agriculture within easy reach by post-cart or light railway. The summer climate, with a mean temperature of 65·9°, is tempered by a somewhat larger rainfall than the other places named, whilst the winter is bright and dry, with bracing air and cold nights; mean temperature, 51·2°. The extremes of shade temperature recorded are 93·7° maximum and 19·2° minimum. Near the town are warm sulphur springs, with a good bathing establishment. In this town and its surroundings are thus to be found excellent residential conditions for the health-seeker of the class which we have in view. Eighty miles to the south-west lies *Barkly East* (5,831 feet), the highest town in the Cape Province, in the midst of a healthy mountainous district, with excellent pasturage.

Kimberley, farther north, on the western border of the Orange Free State Province, and at an elevation of 4,042 feet, is on the main line from the Cape, from which it is distant about 650 miles. It is a large town of about 50,000 inhabitants, 12,000 of them Europeans, with the usual public buildings, club, library, theatre, park and gardens. There is also a large hospital or sanatorium, built by the late Mr. Cecil Rhodes and the De Beers Company, and a model village for the employés of the diamond-mines, the chief industry of the place. With a mean summer temperature of 73·9°, and winter 58·6°, extremes of shade temperature 107° and 27·6°, and a rainfall of 18·26 inches almost entirely in summer, Kimberley is, generally speaking, a warm, bracing climate, particularly dry, bright, and bracing in the winter. The town itself would not be ideal for a chest invalid, especially if of the erethic type; but suburban parts within easy access of the town could be found fairly suitable for those who had business there.

Natal.—Facing the Southern Indian Ocean, this Province possesses a moister and a somewhat warmer climate than either the Cape or the Orange Free State Province, having broadly speaking, twice the rainfall of the latter. On the

whole, the localities within it are adapted for a less robust type of invalid, and for those whose mucous membranes—whether abdominal or thoracic—are irritable. *Durban* is the port of entrance, and is a fairly healthy, warm district. *Pietermaritzburg*, generally spoken of as Maritzburg, containing in 1918 a white population of 18,527, is within seventy miles of Durban, and is the capital of Natal. Situated at an elevation of 2,225 feet above the sea, it is a well-found town, with library, schools, hospital, public gardens, and all the appurtenances of a capital. The mean summer temperature is 69·6°; winter, 60·1°; rainfall, 38 inches, mainly in the summer, when there are heavy storms, the winter months, except September and October (corresponding with our March and April), being fairly dry. Maritzburg is a healthy enough locality for those whose affairs keep them there, but it is somewhat relaxing, being surrounded by hills. For many people with a disposition to bronchial ailments it might be suitable. The surroundings are picturesque and beautiful, and there are many localities in its neighbourhood, such as *Greytown*, suitable for residence.

Estcourt (3,833 feet), another seventy miles farther on from Durban, is a small agricultural town finely situated amidst grazing and stock-breeding country. It has a fine, fairly dry Natal climate, free from dust, and would seem to be well adapted for a country farming life. The little town is an important junction for railway-lines leading to Weenan, Tugela, and Harrismith from the main line to Johannesburg.

The Orange Free State Province.—This comprises the highest tableland of Southern Africa, having a mean altitude between 4,000 and 5,000 feet. Bloemfontein, Harrismith and Ladybrand may be referred to as districts within its borders of special importance from a climatic point of view.

Bloemfontein, 100 miles due east of Kimberley, can be reached by rail from that town or from Port Elizabeth, the nearest route from the coast, or from East London or Durban. The town is situated on a plateau (4,500 feet), with low hills to the north-east and south, and has a population of some 34,000 inhabitants, nearly 15,000 of whom are white. It is a large pleasant town, more of a garden city than either Kimberley, Maritzburg or Johannesburg, and is provided in an ample degree with the usual municipal, educational and

recreatory advantages. Possessed of an admirable climate, Bloemfontein has long been recognised as a health resort for the delicate-chested, its usefulness in this respect only being impaired by its commercial success and extension. As an all-year-round place of residence, it is perhaps one of the choicest of this favoured Province; and there are smaller centres of civilisation within reach, such as *Boshof*, eighty miles distant, where the climate is practically the same, with more immediate country conditions. The district is pastoral, agriculture and cattle-breeding being the main industries. The mean summer temperature is 68·9°; winter, 54·0°; extremes, 94·7° and 20·6° respectively; rainfall, 23·07 inches, distributed over fifty-eight days, forty-four of which occur in the summer months.[9] A marked fall of temperature takes place at night in this, as in all the bright, dry South African climates. This nightly fall is beneficial to health, provided it be duly reckoned for.

Sixty miles due east of Bloemfontein, on the Durban-Bloemfontein line, is the station of *Westminster*. The country around is devoted to agriculture, and divided into farms belonging to English settlers, and on these farms we have known several patients do well in this splendid climate. The following extract from a letter written in July, 1913, from a former patient will be of interest:

"We are here at an altitude of 5,500 feet. What rain we have (on an average 24 inches a year) falls from the middle of September to the end of February, and mostly comes in heavy storms or showers of an inch at a time. Almost every day, therefore, there is a clear blue sky and sunshine from dawn to dusk. In the winter we have no rain, and have had none now since April. The days in winter are warm, the nights bright, crisp, and frosty. It is possible to go about in shirt-sleeves all day long in winter, and sleep out at night. The temperature falls to about 15° of frost in the winter, and rises to about 90° in the shade in summer. Voluntarily or not, it is almost impossible for life out here to be anything but an open-air one, and on account of the lovely climate it is rare to see a window or door shut. A large number of the residents, even when sound, sleep out on the stoep (verandah), which surrounds every house, all houses here being built like bungalows, on the ground floor only."

About thirty miles farther east of Westminster, on the Basutoland border of the Province, is the small town of *Ladybrand*, in the midst of an agricultural district, with a not

very distant view of the Maluti Mountains of Basuto. "A superb climate in winter, especially May to August, and not too hot in summer, as there is usually a slight breeze from the Malutis" (extract from letter of patient—Rev. George Johnson). The little town of *Bethlehem* (5,300 feet), between Ladybrand and Harrismith, on sandstone formation, is also well adapted for reconstituting the pulmonary invalid.

Harrismith, 250 miles from Durban, the nearest port, and 130 miles north-east from Ladybrand on the Kimberley-Durban line, is a sunny, bracing mountain district (5,250 feet), having the Drakenburg range of mountains thirty to fifty miles south and east. The district is grazing and agricultural. Between Ladybrand and Harrismith, on the western side of the Maluti Mountains, lies a rich cereal-farming stretch of country, extending for 100 miles, known as the "Conquered Territory."[8] The healthy townlet of *Ficks* is about half-way on the line of railway, to the south-east of which the fertile country is situated. Harrismith has all the characteristics of a mountain climate in the warmer latitudes, with its bright and brilliant days, always to be followed by cool, and often very cold, nights. It is a climate admirably fitted for persons intrinsically sound, yet with perhaps a strain of pulmonary weakness in their ancestral line.

The Transvaal Province.—The climate of the Transvaal, which in its southern portion ranges at much the same level as the Orange River Province, with which it is continuous, is drier, more dusty, more disturbed by storms, and with greater changes of temperature than that of its southern neighbour. The country is for the most part sandy and dry, with subjacent and extruding granite rocks. Large districts are desiccated in the summer, and violent dust-storms are prevalent. The summer heat is considerable, although the nights are cool. The winters are cold and bracing. The Province has generally been decried for all people with pulmonary delicacy or unsoundness, and yet we have witnessed some very striking examples of benefit derived from a residence in Johannesburg prior to the period of the war.

Johannesburg (5,764 feet) is a city of 260,000 inhabitants, rather more than half of them whites, supplied with all the luxuries of a modern capital, if the price can be paid for them. It is easy of access from the Cape, from which it is distant

957 miles on the main line. Durban is the nearer port, 483 miles *via* Kroonstadt, and it can be reached by a still shorter railway journey from Delagoa Bay *via* Pretoria. Invalids were formerly warned away from Johannesburg, chiefly on account of the dust-storms which prevail from time to time. It is, moreover, an exciting mental atmosphere, with stock-broking and mining industries, wanting in the restful conditions proper to a health resort. Still, there are some who cannot endure a pastoral life, who are inured to business, and can neither live nor make a living without rubbing shoulders and clashing intellects with their fellow-men, and amongst such we have in some cases recommended Johannesburg with great advantage, advising, however, a suburban residence. The size of the town has much extended of late years, and its sanitation has been greatly improved, and is now carefully supervised by able sanitary officers, who by appropriate measures have diminished the dust nuisance. The water-supply is excellent.

The subtropical climate of RHODESIA is not to be recommended for invalids, although *Buluwayo* and *Salisbury* are by no means unhealthy places for those who seek farming and mining pursuits and the sport to be had in such localities. Indeed, the late Mr. Selous,[10] in his interesting book, spoke many years ago of the open grassy downs of Mashonaland as possessed of a delightful climate for the greater part of the year—"a country where European children would grow up with rosy cheeks." The plateau (4,000 to 6,000 feet elevation) was at one time thickly inhabited by a peaceful and a pastoral people.

The Argentine.—For those who have South American connections we may draw attention to the La Cumbre district in the Argentine as one in which we have known consumptives to do well. This district is a high tableland, some 4,000 feet in altitude. Situated in the hills of Cordoba, the mean summer temperature is 70° to 80°, that of winter 40° to 60°. The soil is rich, the air exhilarating, and there is abundant sunshine. La Cumbre itself is a small English colony, and it is often possible to obtain there suitable employment.[11]

The Hill-Stations of India.—These are by no means equal to the others mentioned for the treatment of phthisical cases. The best known is *Darjeeling*, situated at an altitude of about

7,000 feet, among the southern slopes of the Himalayas. The rainfall is, however, 132 inches—very different from the figures recorded at the other high-altitude stations which we have been considering—and during the rainy season it is accordingly very damp. A sanatorium has recently been built in the Kumaon Hills at Lotni, Bhowali, not far from Naini Tal, the summer capital of the United Provinces.

Stations of Medium Elevation (1,000-3,000 Feet).—Among places of lower elevation, we may mention *Görbersdorf*, in Silesia (1,800 feet); *Falkenstein* (1,600 feet) and its neighbourhood, in the Taunus Range, not far from Wiesbaden; *Nordrach* (1,475 feet), *Badenweiler* (1,450 feet), and *St. Blasien* (2,530 feet), all in the Black Forest; *Lausanne* (1,690 feet); *Glion* (2,270 feet); and *Meran* (1,000 feet), in the Austrian Tyrol. At the first two of these, Görbersdorf and Falkenstein, are the well-known sanatoria inseparably connected with the names of their founders, Dr. Brehmer and Dr. Dettweiler, who did so much to introduce the open-air treatment of consumption, though the institution at Falkenstein is no longer used for the treatment of tuberculosis. In these stations of medium elevation, to which we might add others in the *Harz Mountains*, the element of a rarefied atmosphere is wanting, but the air is clear, dry and bracing, and is adapted for certain cases for which the higher resorts are unsuited. At Meran the "grape cure" is conducted in the autumn (see p. 604), the "koumiss cure" in the spring, and baths and inhalations all the year round.

It is only by weighing all the circumstances of each individual case that it can be decided whether on the whole a better result may be hoped for by a sojourn at the higher or the lower stations, and an accurate diagnosis of the physical condition of the patient must ever be the first step towards the solution of the problem.

REFERENCES.

[1] For a more detailed description of these places and of the numerous Swiss summer resorts of high altitude, the reader should refer to—

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[2] *Croonian Lectures on the Hygienic and Climatic Treatment of Chronic Pulmonary Phthisis,* by Hermann Weber, M.D., p. 84. London, 1885.

[3] (1) *The Rocky Mountain Health Resorts,* by Charles Denison, M.D., p. 68. Boston, 1881.

(2) *Analysis of Atmospheric Humidities in the United States,* by Charles Denison, M.D., p. 19. Chicago, 1884.

[4] "What Influence has Climate on the Treatment of Consumption, and how far can Cases be grouped for Treatment in Certain Climates?" by C. Theodore Williams, M.D., *Transactions of the British Congress on Tuberculosis.* London, 1901, vol. iii., p. 14.

[5] *Die Tuberkulosesterblichkeit der Schweiz und die zur Bekämpfung der Tuberkulose daselbst im letzten Jahrzehnt gemachten Anstrengungen,* von Dr. Schmid, Direktor des Schweizerischen Gesundheitsamts in Bern. Tuberculosis. Berlin-Charlottenburg, 1912, vol. xi., p. 352.

[5A] *La cure de Soleil,* par Dr. A. Rollier. Paris et Lausanne, 1914.

[6] For further details concerning the stations in the Rocky Mountains, reference should be made amongst others to the following :

(1) *Rocky Mountain Health Resorts,* by Charles Denison, M.D., Boston, 1881 ; also Dr. Denison's *Climatic Maps of the United States,* 1885.

(2) *Aero-therapeutics, or the Treatment of Lung Diseases by Climate,* by Charles Theodore Williams, M.D. London, 1904.

(3) (*a*) *A Handbook of Medical Climatology,* by S. Edwin Solly, M.D., M.R.C.S. London, 1897. (*b*) "The Health Resorts of the United States," by Edwin Solly, M.D., in Dr. Hale White's *Textbook of Pharmacology and Therapeutics,* p. 965. London, 1901.

(4) *A Handbook of Climatic Treatment,* by William R. Huggard, M.D., p. 257 *et seq.* London, 1906.

[7] *The Place of Climatology in Medicine,* by William Gordon, M.A., M.D., F.R.C.P., p. 31. London, 1913.

[8] *The South and East African Year Book and Guide,* edited annually by A. Samler Brown, F.R.M.S., and G. Gordon Brown, F.R.G.S. London, Edition 1920.

[9] For further details in regard to the climatology of Bloemfontein we must refer to (*a*) the table in the Appendix of the last edition of this work, p. 662 ; and (*b*) *The Climate of the Continent of Africa,* by Alexander Knox, B.A. (Cantab.), F.R.G.S. Cambridge, 1911, p. 433.

[10] *Travel and Adventure in South-East Africa,* by Frederick Courteney Selous, C.M.Z.S., p. 80. London, 1893.

[11] *Sixth Annual Report of the King Edward VII. Sanatorium, Midhurst,* 1912, p. 46.

CHAPTER XLVI

ON CLIMATIC CHANGE IN THE TREATMENT OF PULMONARY TUBERCULOSIS—(*Continued*)

Marine, Maritime, and Inland Climates.

PERHAPS such practical suggestions as we have to offer with regard to the selection of marine and maritime climates, and the inland climates of the plains, will be most convenient for reference if arranged under seasonal headings.

AUTUMN (*September, October, and November*).—At this period of the year the marine and maritime climates are especially suitable, since during the latter part of the season, when the leaves are falling and rotting on the ground, the inland country districts, wherever there are many trees, are eminently unfitted for cases of chest disease or delicacy. Extensive pine districts, if on high ground, form an exception to this general rule. During the first two-thirds of the autumn season the moorland districts of Scotland, Yorkshire, and Devonshire, and such dry localities as *Malvern* and *Tunbridge Wells*, are also well adapted for pulmonary cases.

Seaside resorts are characterised by abundant air space, great purity of air, with relative excess of ozone and of moisture, and an equable temperature, with great freedom from dust. As the season advances the temperature is also relatively warmer, as well as more equable, than in inland districts.[1]

The most material advantages of popular seaside resorts, however, are the abundant accommodation and variety of food which they afford to invalids, with a constant and inexhaustible reservoir of the purest air, and great facilities in the form of dry, level walks, sheltered seats, carriages, bath-chairs, and so forth. For the first half of the autumn season, Northern and East Coast places on our own shores may be selected—*Nairn, Scarborough, Skegness, Clacton, Margate, Clifton-*

ville, *Broadstairs*, *Ramsgate*, *Folkestone* or *Eastbourne*. During the latter part, *Hastings*, *Eastbourne*, *Brighton*, *Ventnor*, *Bournemouth*, *Torquay*, *Newquay*, *Tenby*, *Grange*, etc. The more bracing places should be chosen for those of strumous type, and with no marked tendency to laryngeal or bowel complications; and a residence at these more bracing resorts with an easterly aspect may often be extended far on towards Christmas, or even into January. In hæmorrhagic cases, and those with larynx and bowel complications, the softer climates of *Hastings*, *Ventnor*, *Torquay*, *Weymouth*, *Falmouth* and *Tenby* are as a rule preferable. In cases of nervous, irritable temperaments, with disposition to neuralgia, sleeplessness, and torpidity of the liver, modified sea climates, such as can be obtained a little inland, at *Bournemouth*, *Paignton* and *St. Mary Church*, often agree better.

Of places on the Continent, *Innsbruck*, *Meran* (Austrian Tyrol), or *Montreux*, *Chexbres*, and a few other Swiss resorts, may be advised. These latter places are also convenient for those who contemplate the Riviera or Swiss mountains for the winter, and at either Meran or Montreux the stay may be prolonged into the winter months.

Sea Voyages.—These have enjoyed in the past a great reputation in the treatment of phthisis. The late Dr. Theodore Williams[2], in his Lumleian Lectures comparing the results given by different climates, found that sea voyages yielded in his statistics a larger percentage of recoveries than the Riviera or the home climates, and were second only to the higher altitudes.

We must say at once, however, that, though good results have been undoubtedly obtained in the past in suitable cases from sea voyages, yet it is a form of treatment which is clearly far from ideal. The patient may, under favourable conditions, enjoy abundance of fresh air during the day, but at night, even in fine weather, his cabin can rarely fail to be stuffy, while in stormy seas and with port-holes closed the air may become very foul. A voyage, therefore, is by no means a perfect open-air life, as is sometimes hastily imagined. To this drawback we must add the possibility of sea-sickness, the changes of temperature to which, as the voyage proceeds, the patient is exposed, together with the enervating influence of the moisture and heat during the passage through the tropics.

Further, should the patient become ill, or have a recrudescence of his malady, it will not be easy to obtain for him the requisite nursing or proper food. It is clear, therefore, that the scope of the ordinary sea voyage is a restricted one. Whether it will ever be possible, as has been suggested, to have a vessel arranged as a floating sanatorium on a sufficiently ample and luxurious scale for the treatment of patients at some ideal locality on the high seas is a matter, perhaps, worthy of consideration.[3] The idea would not be difficult of realisation to a man of ample means, with a well-equipped private yacht.

The sea climate contrasts with that of the mountain valley in excessive moisture, comparative equability of temperature, and high and variable atmospheric pressure; in freedom from organic and inorganic dust they resemble each other. And it is instructive to note that, allowance being made for individual peculiarities, the same class of cases do well under both conditions.

Patients with active tuberculous disease should be strictly forbidden to take a sea voyage, the only exceptions being in the case of those who have friends and better home surroundings at their proposed destination. Cases of chest delicacy, on the other hand, especially in those with overworked nervous systems, quiescent disease with defective sanguification, and early cases associated with enlarged glands—the so-called "scrofulous phthisis" of earlier writers—do well on a voyage. But even for these the voyage should not be countenanced unless the patient have been already carefully observed for some time at home, to be certain that the disease shows no activity, or, better still, have already spent some time in a sanatorium where he has been carefully watched, and instructed in regard to his personal hygiene and mode of life. Hæmorrhagic cases and those complicated with dyspepsia or diarrhœa are not adapted for sea voyages.

A fundamental question to ask a patient for whom one is contemplating the recommendation of a voyage is, whether he is a good sailor and eats and sleeps well on board ship. Unless a fairly confident affirmative answer can be given to these questions, it will be best not to advise—in the first instance, at least—any sea trip longer than to the Canaries, or perhaps the Cape. Unless, also, the voyage can be taken

with first-class accommodation and with every possible luxury, including a deck-cabin, so as to insure as much ventilation as possible, it should not be advised. Moreover, it is important that some definite plan be agreed upon beforehand as to route and destination. A certain steadiness of purpose in this respect is essential to those who would profit by their temporary expatriation. Patients who go to distant regions in search of health, and find it on the way, frequently by the time of their arrival have forgotten their original purpose, and scamper about over various climatic areas in a manner calculated entirely to thwart the object with which their journey was designed.

We may here again refer to the restrictions placed by the Governments of Australia and New Zealand, of Canada, the United States, and the Union of South Africa upon entry into their territories of patients suffering from tuberculosis, and the advisability of communicating with the representatives in London of the various Governments concerned before the patient leaves England, so that he may not find that he is precluded, as an unsuitable case, from landing when he reaches his destination (see p. 636).

The latter part of the autumn, as soon as the equinoctial gales are over, is the best time for commencing a long sea voyage. Shorter trips are advantageously taken in the earlier part of the season.

In former editions of this work the long sea voyage to Australasia by sailing-vessels round the Cape was considered. But at the present day well-found vessels of the kind are not available, and only by special arrangement with a few trading-vessels still remaining can this restful and health-giving voyage be entertained by those thorough sons of the sea who can enjoy it. It will be taken for granted, therefore, that the Orient, P. and O., R.M.S.P., or some other great line of steamers, will be made use of for the voyage.

Having reached *Australia* patients should only remain at Melbourne the briefest possible time, the climate there being most unsuitable for chest diseases; nor is that of Sydney very suitable at the time of year calculated for arrival.

Hobart, in Tasmania, is easy of access from Melbourne, either by steamer direct, or to Launceston and thence by rail. The climate of Hobart is perfect from November to May, and

the hotels, boarding-houses, and apartments of the neighbourhood are good. Children do well there, and a patient with arrested disease who desired to remain at the antipodes during our worst months, with the view of returning home about June, could not do better than spend the interim at Hobart. In the case of robust and independent patients with little or no actual disease, but who, invigorated by their journey out, desire simply to lead a more outdoor life than is possible in their own country, certain parts of Australia are suitable. For such the neighbourhood of *Bendigo*, north of the Victorian range of mountains, the *Riverina*, in New South Wales, or *Warwick* and the *Darling Downs*, in Queensland, afford ample fields for pastoral life. Of late the *Blue Mountains*, not far from Sydney, have acquired a reputation as a health resort for sufferers from consumption, and yearly attract many Australian patients.

It is often of the highest importance for consolidating a cure to spend at least two winters away, and the patient can for this purpose remain at Hobart until May, and then visit Sydney or the interior of Victoria, Queensland, or New South Wales; or an outdoor sheep-farming life may be led in Northern or Western Victoria, except for the two hottest months of December and January, which should be spent in Tasmania or New Zealand.

The climate of *New Zealand* may be said, generally speaking, to be an improvement, for chest invalids, upon Southern England, having more sunshine and warmth, and greater possibilities of outdoor life, but with a decidedly stormy and variable climate, even in summer the nights being often very cold. The choicest parts of New Zealand are, perhaps, *Napier* in Hawkes Bay, North Island, and *Nelson* in Tasman Bay, South Island. These stations are warm, fairly equable, and afford good accommodation. The interior of Otago is spoken well of, but it is colder than the other places named.

A plan of combining a shorter voyage with residence away from England for twelve months would be by P. and O. steamer, so timed as to reach *Calcutta* after Christmas, spending January and February there, and then proceeding to *Darjeeling*, where the rest of the year, from March to November, might be passed, returning, after making a further stay at Calcutta, by the Riviera gradually homewards.

By one or other of the plans just suggested, the advantage gained by a long sea voyage may be still further improved, one or two winters being escaped, and the patient returning home at the beginning of the second or third summer. In favourable cases, after a year or two devoted to recovery, some suitable locality may be fixed upon for permanent residence.

WINTER (*December, January and February*).—For those patients with pulmonary tubercle in an early and quiescent stage who cannot go abroad in the winter, there are still English places fairly well adapted to meet their requirements —viz., the maximum enjoyment of air and exercise out of doors. In cases in which only a period of three months can be spent abroad, it is far preferable to select the three spring than the three winter months; for with the setting in of the winter frosts there is no longer the same harmful influence to be dreaded in inland leafy districts as in the late autumn. Those patients who have homes in our inland counties situated on dry, porous soils, with sunny exposure and fairly protected from cold winds, may do well there, and many invalids, with a short change in the autumn and spring, will do best thus to spend the winter months at their own homes. After years of weary travel, patients not infrequently find their immunity, and can then remain in their own home district, provided conditions are fairly favourable.

Hastings and *St. Leonards*, *Ventnor*, *Bournemouth*, *Teignmouth*, *Torquay*, *Falmouth*, *Penzance* and *Tenby* (south side), may be selected by those not well situated at home; of these places the air of Teignmouth, Torquay, Falmouth, and Tenby, is softer than at the others, and more suitable for cases with dry, harsh skins and irritable mucous membranes. It is to be remembered that, whereas at Hastings, St. Leonards, and Ventnor the coast residences should be chosen, and the uplands only in special instances, at Torquay, on the other hand, the quay residences are cold, damp and misty from defective sun exposure and imperfect circulation of air, whilst the terraces on the slopes are comparatively warm, sunny and dry. *St. Mary Church*, a suburb of Torquay, suits some people better, being more withdrawn from the sea.

Bournemouth is a sea climate, much modified by the extensive moorlands behind it and the pine growths, amidst which

many of the best houses are placed, although of late years the spread of the town has, unfortunately, led to the cutting down of many of these trees. The habitations are more isolated and stand farther back from the sea than at other places, and these facts render the climate a happy mixture of marine and moorland, suitable for many people with whom a more purely sea air disagrees. An asthmatic or bronchial element would suggest Bournemouth in preference to other places. In point of warmth Bournemouth is not to be preferred to any of the other South-Coast resorts; like most other large places, however, such as Torquay and Hastings, it possesses many sheltered nooks well known to resident practitioners—indeed, these places may be said to be "full of climates!"

The Riviera, that narrow strip of land bordering the Mediterranean, and bounded and protected on the north by the Maritime Alps, extends from Hyères eastwards. The portion between Hyères and Genoa is known as the Western Riviera, or Riviera di Ponente, and constitutes the Riviera proper; most of the well-known health resorts are situated in it. From Genoa to Spezzia, or even to Leghorn, runs the Eastern or Levantine Riviera. This at the present time is less well known, and in pre-war times was less expensive; but its climate, though moister and possibly somewhat colder, does not otherwise differ materially from that of its western rival.

The Riviera climates have in common the advantages of warmth, brilliancy, and comparative dryness. They are easily accessible, and the larger resorts possess excellent accommodation. They differ among themselves in exposure to wind, especially the cold north-west wind or mistral, and also in the degree of prevalence of dust. Certain of these points are brought out by the meteorological data which we have collected (see table, p. 643). From these it will be seen that the mean temperature for the six winter months (November to April) at Nice, Cannes, Mentone, and San Remo varies between 50·0° and 52·0°, that of London (Regent's Park) being 41·9°, whilst Hastings, Bournemouth, and Torquay yield means of 42·5°, 43·8°, and 45·1° respectively. Records are wanting to enable us to compare accurately the amount of sunshine on the Riviera with that of other stations, but that it is greatly in excess of that enjoyed at English resorts,

and, owing to the dryness of atmosphere, of a brilliancy rarely seen with us, may be accepted.

The rainfall during the winter months may be taken at about 17 inches (Nice, 15·5 inches; Cannes, 19·1 inches), which is somewhat in excess of that at Regent's Park (11·31 inches) and of Hastings (14·50 inches), but not very different from that of Bournemouth (15·93 inches) and of Torquay (17·37 inches). Nevertheless, owing to the rain coming rather in storms, and the sky then clearing, the number of rainy days on the Riviera is materially less than in our own climate. Thus, at Nice, Cannes, and Mentone, there were respectively 43, 43, and 47 rainy days during the six winter months, while in London (at Regent's Park), Torquay, and Hastings they amounted to 83, 90, and 101 during the same period. The dryness of the atmosphere is further indicated by the records of mean relative humidity at 9 a.m., Dr. Marcet's figures for six winters at Cannes yielding a percentage of 73, as compared with 82 and 84 for Torquay and Regent's Park respectively.

Such are the more important characters of the climate of this favoured region, and experience shows that one or other of the resorts along its shores is generally suited for those cases for whom the elevated stations are not desirable. Phthisical patients with poor general vitality will do best to spend the first winter, at all events, in the Riviera. In some cases, also, in which the necessity for change of climate occurs when cold weather has already set in, it is more prudent to select one of the warmer resorts, with the view, perhaps, of higher ground in the ensuing autumn.

There are, moreover, some early-stage cases of phthisis, complicated with threatened or actual laryngeal or bowel trouble, in which the prognosis is grave, and in which the cold air of the mountains is not to be advised. In such cases, provided the patient can go abroad with every comfort and accompanied by his family or nearest friends, relief of symptoms and lessened activity of progress may be effected by a visit to one of the sheltered Riviera resorts.

Children and young adults, especially girls, with a delicate family history, who have had acute bronchial or pulmonary affections, may, by one or two winters spent in the Riviera, completely and permanently recover. To elderly people,

again, with phthisis, in whose tissues senile decay has already commenced, these warmer climates are admirably adapted.

A most important point to be borne in mind by those who send invalids to the Riviera is that warm woollen underclothing should be enjoined, and that they should be especially warned to be within doors half an hour before sundown, a fall of several degrees of temperature taking place abruptly at this time. The patient should accordingly never run the risk of being belated out of doors without special wraps, although, properly clothed, he may often with advantage go out again later in the evening.

Of the Riviera resorts, the following, as we pass from Hyères eastwards, are the more important:

Hyères.—The west end of Hyères is the best part, its elevation permitting of superior drainage. Somewhat removed from the sea, Hyères enjoys a slightly modified sea climate, and is partially protected from winds by the chain of islands parallel to, and two or three miles distant from the shore. The climate is accordingly more soothing than many of the Riviera stations, and excitable people of the "erethic" type will probably do better here than elsewhere. Some forms of asthma, bronchitis, and emphysema, are also benefited. From November to February the climate is at its best, and during this period may be recommended for cases of advanced but quiescent tubercle, and those complicated by albuminuria. In February and March the mistral is sometimes severely felt.

Costebelle, two miles south of Hyères, on the Hermitage Hill, 200 feet above the town, is quieter and less dusty. It possesses excellent hotel accommodation, and may be recommended for the same class of cases for which Hyères is suited. There are excellent golf-links in the district.

Cannes.—For prolonged residence Cannes affords the most varied attractions, perhaps, of any of the Riviera stations. Yet in the pleasures of its social life lurks a danger to the invalid against which he must be on his guard; for he must ever remember that his object is to regain his health, and that this can only be effected by rigidly ordering his life aright under proper medical supervision, by keeping early hours, and by eschewing for the most part the numerous gaieties for which the season at Cannes is famous. The upland parts of the town, away from the shore, are best suited for residence.

The climate (see table, p. 643) is decidedly bracing and brilliant, though somewhat changeable. Cases of quiescent phthisis, of strumous type, with lax secreting membranes and non-pyrexial sweatings; cases of anæmia, senile forms of bronchitis, emphysema, asthma; and persons, both young and old, recovering from acute chest disease, do well here. For patients, on the other hand, of highly nervous temperament, with a disposition to sleeplessness, Cannes is not so well suited. In this large district there are many climates, and it is safest to take local advice as to any precise locality for prolonged residence.

Cimiez, a suburb of *Nice*, is well suited for cases of asthma, for neuralgic patients, and bad sleepers. It is less liable to variable winds than the town of Nice. For elderly people Nice has the advantage of level walks and promenades and town life, with the brilliant Riviera sunshine; and there are many sheltered parts in the Carabaçel quarter, in which more delicate patients will find protection from cold winds.

Beaulieu, situated between Nice and Monte Carlo, is wonderfully warm and sheltered, and is admirably suited for patients needing such a winter climate.

Monte Carlo and Les Moulins, in the Eastern Bay of the principality of Monaco, are amongst the choicest spots of the Riviera for residence, the dwellings being between 200 and 300 feet above the sea-level, well protected from mistral by mountains behind, yet with abundant air circulation. These places are to be recommended, however, only for wealthy people in limited numbers. *Cap d'Ail La Turbie*, in the neighbourhood, where there is a good hotel, is well adapted for pulmonary convalescents without any declared disease.

Mentone.—This resort, especially the East Bay, is warmer and more sheltered than the other stations, with the exception of Beaulieu, and yet has a good circulation of air. Many real invalids accordingly make their winter-quarters here. The mean temperature for the six winter months may be taken as 52° (Bennett). A glance at the table on p. 643 will show that this is slightly higher than the corresponding figures for San Remo, Cannes, and Nice. Mentone is well adapted for residence in cases of chronic bronchial catarrh, gouty bronchitis, and phthisis with albuminuria. Asthmatics frequently also do well in Mentone. Cases of somewhat advanced phthisis find

this locality soothing; and persons who lead, from cardiac diseases or other causes, lives of enforced inactivity will often profit by sojourn here. Patients in whom hæmorrhage is a marked feature are not well suited for this very marine climate, a more inland locality being better adapted to them. The West Bay of Mentone is somewhat fresher and more bracing than the East, but the sanitation of both districts is equally good. *Cap Martin*, a bracing peninsular suburb of Mentone, with an excellent hotel, is well adapted for wealthy convalescent patients.

San Remo is a charming station, which has grown steadily in popularity. It occupies a sheltered position some eight miles east of Bordighera, itself a growing health resort. Its winter climate (see table, p. 643) resembles that of Mentone, although, as we have seen, it is a trifle colder and not so well protected from wind. It is admirably adapted for fairly quiescent cases of consumption, especially those associated with a languid and feeble circulation. Cases of heart disease and emphysema do well at this resort, where a long stretch of level walk extends along the front and through the newer part of the town.

In addition to the stations which we have named, there are many other places worthy of note along the northern coast of the Mediterranean. Such are *Alassio* and *Pegli*, on the Western Riviera; *Nervi*, *Portofino*, *Rapallo*, and others on the Eastern or Levantine Riviera, the latter not as yet fashionable, and consequently, before the war, more moderate in price. *Algeciras* and *Malaga*, on the south coast of Spain, and *Monte Estoril* in Portugal, not far from Lisbon, may also be mentioned. All have their merits, and at Nervi and Portofino and Algeciras good hotel accommodation may be obtained.

On the way to the Riviera, Arcachon, Biarritz, and Pau may be mentioned. *Arcachon*, in the district of the Landes, some thirty miles south-west of Bordeaux, although colder than the more southern stations, is well adapted for many cases of arrested pulmonary tuberculosis with excitable rather than depressed nervous system. The winter villas amongst the pines are the most suitable, and riding exercise can be enjoyed amongst the interminable pine forests, which afford consider-

able shelter from the spring winds. Some cases of chronic phthisis with excessive secretion do well here. The climate is also well adapted for asthma.

Biarritz, on the shores of the Bay of Biscay, some sixteen miles from the Spanish border, possesses a highly marine climate. It is suitable in the late autumn and *early* winter and later spring months for threatened cases of consumption, especially in children for whom the bracing effect of the Atlantic breezes may be desired. It is too much exposed to wind for most cases of declared disease. People with a disposition to neuralgia do not do well here.

Pau, an inland station in the Basses-Pyrénées, about sixty miles south-east of Biarritz, possesses a winter climate some five degrees colder than that of the Riviera; its rainfall and humidity are also greater. The atmosphere is, however, very still, and winds are but little prevalent. Cases of chronic bronchial and laryngeal catarrh, and some elderly patients who do not flourish at the Riviera climates, are better here. It is a sedative climate, with magnificent surroundings and sheltered walks and drives, and many social attractions.

Algiers, on the southern shores of the Mediterranean, has a mean winter temperature of 58·7° (see table, p. 643), between 8° and 9° higher than that of Cannes or Nice, but its climate is moister than that of the Riviera resorts. The rainfall, 23 inches from November to April inclusive, is also greater, whilst the days on which rain falls are nearly double, eighty-four (almost identical with that of London), as opposed to forty-three at Nice and at Cannes. Algiers is, however, but little affected by mistral, and rarely visited during the winter and spring months by the dusty south wind or sirocco.

The climate is suitable for cases of chronic bronchitis, emphysema, quiescent phthisis, even if somewhat advanced, and for some cases of asthma. For all these affections the Mustapha Supérieur quarter of Algiers is to be selected.

Egypt.—The warm, very dry winter climate of Eygpt, with its pure desert air, brilliant sunshine, low degree of humidity, and but scanty rainfall—factors well brought out by the meteorological data relative to Helwân, which we have incorporated in the table on p. 643—would seem to offer consider-

able advantages to the consumptive, and certain cases find relief there. Egypt is perhaps best suited for patients with quiescent disease but continuing secretion, which tends to diminish and dry up under the favourable climatic conditions. It is, however, less easy at the Egyptian resorts than at other places which we have considered to insure that continuous medical supervision which is so essential for successful treatment, or for the patient to carry out the régime laid down for him, even if so minded. Moreover, experience has shown that the majority of cases of early phthisis, provided they have sufficient vitality, do better in a more bracing climate. Still, we have known cases do well in Egypt, and their lesions to remain arrested after spending some winters there. This climate is more specially adapted for the chronic bronchitic and asthmatic invalid.

Those who go to Egypt in search of health should eschew Cairo, with its gaieties and dust, and go to *Mena House* or *Helwân*, not far from Cairo, or to *Luxor* or *Assouan*,[4] on the banks of the Nile farther south. Dahabieh excursions up the Nile may be suggested as an interesting change for the wealthy, although under no circumstances can a winter in Egypt be passed without considerable expense.

The Egyptian season ends in April, when the weather becomes too warm. Patients should not then hurry back directly to England, very possibly to find there the winter which they have successfully eluded so far, but should be advised to stay at some intermediate station, perhaps in Sicily, at one of the Riviera resorts, or on the Italian lakes, at, for example, *Varese* or *Lugano*, until the end of May, when the return home may be sanctioned. *Meran* in the Austrian Tyrol or *Vernet-les-Bains* in the Pyrenees may also be recommended. Another feasible plan is to take the P. and O. boat to Gibraltar, and cross over to *Algeciras*, where excellent accommodation can be found at the hotel.

Madeira and its principal town, *Funchal*, enjoy a warm, moist, and equable climate, abundant sunshine, and freedom from dust.

The temperature records, as will be seen from the table on p. 643, correspond closely with those of Algiers. Thus, the mean winter temperature at Funchal is 59·9°, as compared

with 58·7° at Algiers; while the range is slightly less (12·7°, as against 13°). The winter rainfall is, however, smaller, and the number of rainy days materially less (fifty-three, as opposed to eighty-four). As compared with the English stations, the mean winter temperature at Funchal is some 15° higher than that of Torquay, and 18° higher than that of London (Regent's Park). The winter rainfall is considerably greater, but the number of rainy days materially less (fifty-three as opposed to ninety at Torquay, one hundred and one at Hastings).

A noticeable feature of the climate is its equability and the slight difference between the mean temperatures throughout the year. At Funchal the mean for the six winter months (November to April) is 59·9°; for the six summer months (May to October), 67·7°. In former times when equability and warmth were regarded as of the first importance in the treatment of phthisis, Madeira, with its warm marine climate, beautiful vegetation, and comparatively easy accessibility, was much resorted to. The patients, however, from the Brompton Hospital who were sent to winter there yielded unsatisfactory results, only three out of the twenty deriving benefit, and the statistics published by the late Dr. Theodore Williams[5] in his Lettsomian Lectures in regard to his Madeira patients were also unfavourable. At the present time opinion is in favour of a colder and more stimulating climate, provided the patient can respond, as leading more readily to the arrest of disease.

In cases of emphysema, and of phthisis with a good deal of attendant bronchitis and emphysema, and especially in those of older years in whom the disease has supervened upon long-standing winter cough, the climate of Madeira will be found well adapted for residence. Patients with irritable cough and but little expectoration, or with laryngeal trouble, provided it be not too advanced, may also find relief here, such cases being as a rule unsuitable for high altitudes. For many bronchial and some asthmatic affections, particularly those associated with renal unsoundness, Madeira is peculiarly suited.

At different elevations from Funchal, up to a height of 2,000 feet, more bracing accommodation can be obtained than in Funchal itself, so that, owing to the coolness of the summer the patient need not hurriedly leave the island the moment the winter has ended, but is enabled to remain in comfort until

May or early June, when he can more safely return to England. The absence of dust and of mosquitoes is especially to be emphasised as a feature of this climate.

The Canary Islands possess a climate which resembles that of Madeira, but is somewhat warmer, less moist, and more dusty. It may be recommended for similar cases. *Oratava*, in the island of Teneriffe, or *Las Palmas*, in Grand Canary, are suitable for a lengthened stay; and the latter especially so in cases in which a rheumatic element is present.

We have observed *Santa Cruz*, at the foot of Oratava, to be especially suitable for asthmatic cases.

California.—The climate of that portion of the State of California which lies south of Point Conception between the mountains and the Pacific, is characterised by mildness, the winters being warm and spring-like, with abundant sunshine, whilst the summers are cool. The rainfall is a small one, but the humidity, owing to the influence of the ocean, is not particularly low, and fogs in the morning and evening are by no means infrequent.

The climate at the various resorts will be found to differ somewhat according to local conditions, and especially with their greater or less proximity to the coast; but, taking the meteorological data of the city of *Los Angeles* as an example, we may note that in point of temperature the records (see p. 643) show a climate which, so far as this feature is concerned, closely approximates to that of Madeira. Thus, at Los Angeles the mean winter temperature is 57·1°, at Funchal, 59·9°; the mean summer temperatures being in each case 67·7°. The daily range—in winter, 21·2° at Los Angeles—is, however, considerably greater. The brilliant sunshine enjoyed by the Californian resorts is also demonstrated by the records, which show that as much as 3,219 hours were recorded during the year, 1,457 of which occurred in winter. For comparison, we may add that the yearly totals at Bournemouth, Torquay, and Hastings amount to 1,717, 1,731, and 1,783 hours; and for the six winter months, 572, 568, and 580 respectively. The rainfall at Los Angeles is only 15·6 inches, and the number of rainy days throughout the year not more than forty. A glance at the table on p. 643, and at the more detailed tables printed in the appendix to the last edition of this work, will show

how favourably these figures compare with those of other stations.

With a genial climate of this nature, the health resorts of Southern California will be found to afford winter-quarters suitable for those phthisical patients with low vitality, or with disease too advanced for the American altitudes, or for whom those climates are for other reasons undesirable. Among the resorts we may mention *San Diego* and *Santa Barbara*, on the Pacific coast, *Pasadena*, some twenty miles inland, not far from Los Angeles, and *Sierra Madre*, in the same region. At *Monrovia*, sixteen miles from Los Angeles, there is a well-known private sanatorium.

These stations are too far removed from England for temporary resorts, but are available for American invalids, some of whom after their recovery may decide to make their permanent residence in such localities, and perhaps find light and satisfactory outdoor occupation in connection with fruit-farming and the cultivation of oranges and lemons, for which California is famed.[6]

Spring (*March, April, and May*).—Our islands can boast of but few localities suitable for chest invalids during spring. There are, however, numerous nooks and corners with sunny exposure and protection from cold winds, where on porous soil and at a moderate elevation residences exist or might be built, singly or in small groups, suitable for invalids who are unwilling or unable to go farther. At some of our seaside places, such as *Bournemouth*, the *Isle of Wight*, *Torquay*, *St. Mary Church*, *Tenby* or *Grange*, sheltered spots can be found. *Bridge of Allan*, by Stirling, N.B., is a spring station of considerable merit, and here, on the southern slope of the hill, protected from the north and east, are houses and a well-found hydropathic establishment with sheltered walks.

Throughout the Continent the same difficulty presents itself to a greater or less degree with regard to the avoidance of irritating cold spring winds. With the exception, however, of this occasional drawback, most felt during February and March, the whole Riviera is at its best during the spring season. Parts of *Mentone*, *Beaulieu*, *Monte Carlo*, *Bordighera*, *San Remo*, and some sheltered portions of *Hyères*, are the most protected; but it is, perhaps, scarcely wise, on

account of occasional cold winds, to shift quarters that are otherwise suitable. *Grasse* (1,000 feet), a bracing hill suburb of Cannes, nine miles from the coast, is a good late spring resort. *Les Avants*, above Montreux, is also protected.

Many patients who have spent the winter at the Riviera are tempted at this season to move on to Florence (where, however, the spring winds are much felt) or to Rome. In the case of pulmonary invalids this is not to be sanctioned.

Madeira, the *Canaries*, and *Algiers* are not affected by severe spring winds; but the latter place becomes too warm for residence in the later spring. *Arcachon* and *Pau* are fairly good spring climates. At this season of the year a voyage to the *West Indies* may often be recommended for convalescents from acute chest diseases.

SUMMER (*June, July, and August*).—In the summer months the travelled and tired invalid will generally do best to return to his home and familiar haunts, friends, and diet. June is a favourable month for visiting certain health resorts and baths, such as *Ems*, *Aix-les-Bains*, *Aix-la-Chapelle*, *Allevard-les-Bains*, *Royat*, *Mont Dore*, *Eaux Bonnes*, but chiefly for the treatment of special throat or other symptoms, to be incidentally alluded to elsewhere.

After a period of treatment, such as we have sketched out, the patient may have regained his health sufficiently to allow of his return to work. We have pointed out in a former chapter (p. 622) that it is better for him, if he has been a city dweller before, to alter the environment and to live in future in the country or some small county town. If, however, his occupation must be carried out in a large city, then he should live out of town or in some healthy suburb. For those whose work lies in London, we may recommend such localities as *Hampstead*, *Highgate*, *Golder's Green* or *Hendon*. In the case of those who can live farther afield, the neighbourhood of *Croydon*, *Purley*, *Woldingham*, *Oxted* and *Tamworth* may be recommended amongst others, or the *Chilterns* in the neighbourhood of *Chorley Wood*, *Great Missenden*, and *Amersham*. We have also known not a few patients derive benefit from living at *Westcliff-on-Sea* or *Southend*, where long week-ends may be enjoyed, and whence there is a good train service to London.

In choosing a residence regard must be had to dryness of locality, abundant sunshine, and protection from prevalent winds. Trees must not be in too close proximity to the house.

REFERENCES.

[1] For a consideration of the meteorological data bearing on these differences, see the article on "The Climate of the Midland Counties," by P. Horton-Smith (Hartley), M.D., in *The Climate and Baths of Great Britain,* vol. ii., p. 119. London, 1902.

[2] See "What Influence has Climate on the Treatment of Consumption and how far can Cases be Grouped for Treatment in Certain Climates?" by C. Theodore Williams, M.D., *Transactions of the British Congress on Tuberculosis,* vol. iii., p. 14. London, 1901.

[3] "Treatment of Tuberculosis at Sea," *The Lancet,* 1909, vol. i., p. 1188.

[4] For a consideration of the differences in climate between these four stations see the meteorological data given in "A Clinical Lecture on Mitral Regurgitation," by P. Horton-Smith (Hartley), M.D., *The Clinical Journal,* February 12, 1902, p. 271.

[5] *The Influence of Climate in the Prevention and Treatment of Pulmonary Consumption,* by Charles Theodore Williams, M.D. London, 1877.

[6] For a more detailed description of the climate and of the health resorts of California we must refer the reader to—

(1) "The Health Resorts of the United States," by S. Edwin Solly, M.D., in Dr. Hale White's *Textbook of Pharmacology and Therapeutics,* p. 958. London, 1901.

(2) *A Handbook of Climatic Treatment,* by William R. Huggard, M.D., p. 266. London, 1906.

CHAPTER XLVII

TREATMENT OF PULMONARY TUBERCULOSIS—(*Continued*)

Acute First-Stage Cases—Active Softening and Formation of Cavities—Summary.

Acute First Stage.—In the active periods of the more acute forms of tuberculosis, so long as there are signs of recent consolidation, with maintained pyrexia, rapid pulse, troublesome cough and expectoration, the latter perhaps blood-stained, patients require complete rest, careful nursing, and treatment under open-air conditions, until the acute period of the illness subsides. At this time the patient is receiving discharges of bacillary toxines into his blood; his antitoxic reaction is strained, and his phagocytic powers overwhelmed by the heavy doses of the poison. The hurried action of the heart and breathing and frequent cough all bear testimony to the necessity for quietude; any movement or change from passive recumbency still further quickens the breathing, raises the blood-pressure, and helps to waft into the blood-current the poisons which are being locally elaborated.

The diet should be nutritious and plentiful, and two or three pints of milk should be given each day, in addition to such light solid food as can be borne; for it generally happens that the patient has emaciated and is considerably below his normal height-weight ratio. Stimulants, unless the appetite fails, are as a rule better avoided. The secretions must be cleared, and in cases where the circulation is much quickened and the skin dry and hot an effervescing saline mixture may be given every four or six hours. Presently an alkaline mixture should be substituted, and to it may be added, when the temperature has abated, hypophosphite of soda, to be taken three times in the twenty-four hours. When pain is

complained of, some epispastic solution should be painted over the part affected, or in acute cases two or three leeches applied.

The night perspirations at this early period of the disease are rarely of a severe character, and especially is this the case since the introduction of the open-air régime, and it is wise as a rule not to check them by any drug treatment. If the temperature at night range high, it may be moderated by tepid sponging and quinine, and half a grain of opium may be given if the cough causes restlessness. In other cases a cachet containing 7 grs. of aspirin and 3 grs. of phenazone will check the fever and soothe the erethism with which it is attended. Under such treatment the pyrexia will in favourable cases subside, or at least be reduced to a moderate evening rise of temperature to 99° or 100°, which may persist for some considerable time.

Restraint of the movements of the chest over the affected area by lung splints and other appliances has been suggested; but the normally slight expansile movements of the apices become further restricted in disease without mechanical aid, and the object of rest to the part is sufficiently secured by insisting on that freedom from effort or exercise which shall secure the most complete quietude possible to the respiratory and circulatory systems during the first febrile period of the disease.

It is of great importance to relieve disturbing night cough, a symptom distressing to the patient, and destructive of that functional rest of the lung which we are desirous to secure. The following mixture may be usefully prescribed for this symptom: Nepenthe ʒi., Codeinæ gr. i., Syrupi Chloral Hydratis ʒvi., Mucilaginis Acaciæ, ℥i.ss., Syr. Pruni Virginianæ ℥ss., Aquam Chloroformi ad ℥vi., of which one tablespoonful should be sipped from the spoon at bedtime, and half a dose similarly taken once or twice in the night.

It will often be observed that the cough remains troublesome, while the pulmonary signs are greatly improving, and all secretion sounds rapidly drying up. This irritable cough, which is so frequently attendant upon the subsidence of pulmonary disease, should be checked by the patients themselves as much as possible. This they can do to a great extent, but they may be assisted, if necessary, to secure rest at night by

some such mixture as we have mentioned above, or with the help of one or two morphine and ipecacuanha lozenges. The morning cough in these cases, and, indeed, in many others, is the most troublesome. It is, however, the natural consequence of a good night's rest, and should never be checked by a sedative, since the matters suitable only for elimination, if retained, considerably impede respiration, become highly irritating, and set up further inflammatory and specific trouble in the lungs. A cup of hot coffee, tea or milk, taken before rising, will greatly facilitate expectoration. If this does not suffice, a small dose of ether and ammonia should be given, or if the phlegm be viscid and difficult to cough up, a mixture containing Ammonium Carbonate gr. iii., Sodium Bicarbonate gr. vii., Spirit of Chloroform ♏x., distilled water to half an ounce, to be taken in an equal quantity of hot water on first waking. The old-fashioned remedy of rum and milk taken early in the morning is also useful for this purpose; a dessert-spoonful of rum to a claret glass of warm milk being sufficient.

On the subsidence of fever, or in more serious cases its abatement to an evening rise of temperature only, cod-liver oil may be given, and some tonic containing arsenic, iron, mineral acid or alkaline bitter, as the general and particular features of the case may suggest. In many cases such tonics are not indicated, and in any case it is rarely necessary to give more than two doses a day, at times carefully specified with regard to the meals, according to the drugs prescribed.

The question of allowing the patient up and of permitting exercise may now be considered; but as a rule no relaxation should be permitted until both morning and evening temperatures have been normal for some two or three weeks. He may then be promoted to a couch on the balcony, or in the Liegehalle or garden shelter, and finally the effect of graduated exercise will be observed. Perhaps the best way to begin is to allow a quiet walk of five minutes out of the hour, until the required amount, be it ten, fifteen, twenty, or more minutes of daily exercise, has accumulated. The patient is now in a condition suitable for removal to a sanatorium, where he will have the advantage of that constant medical supervision which is so desirable in the treatment of his case, and especially necessary in graduating the daily

amount of exercise which may be safely permitted (see p. 606).

The Uses and Administration of Cod-Liver Oil.—The advocacy of the use of cod-liver oil in phthisis by the late Drs. C. J. B. Williams and Hughes Bennett gained for this remedy a recognised value second to none other in the treatment of the disease, and from the statistics of the Brompton Hospital, published in the *First Medical Report*[1] (1849), as well as from the results obtained in private cases and recorded by Dr. Theodore Williams,[2] there can be no doubt that at the time of its introduction, and as an adjuvant to the methods of treatment then in vogue, it was of great value. At the present day, when, by a more bracing régime and a more generous diet, the tissue metabolism of the patient is already being stimulated to the full, its value is less marked. It is, however, still indicated when the patient is below weight and does not respond well to ordinary treatment, and it is especially useful among the poor, when the diet is not all that can be desired.

Cod-liver oil has been sometimes regarded rather as a food than a medicine, and its easy assimilation and absorption render it no doubt a valuable nutrient. In the recent shortage of butter and cream and other fatty foods, cod-liver oil alone or combined with malt extract has been a useful resource. In some way, also, cod-liver oil appears to affect favourably the patient's metabolism in a manner that cannot be altogether accounted for by the quantity taken, assuming it to be only a food—one to four teaspoonfuls two or three times a day being the average dose. The observations of Dr. Wells,[3] which have since been confirmed, suggest that in addition to its easy absorption, it possesses the power of "increasing the absorption of the other fats of the foods to a marked degree," and also leads to a diminished excretion of nitrogen, and thus to an increased storing up of proteid within the body. It would seem also from the experiments of Drs. Williams and Forsyth[4] that the unsaturated fatty acids, of which the oil is almost entirely composed, tend to produce a disintegration of the waxy envelope which surrounds the tubercle bacillus, thus possibly leading to its more easy destruction in the blood and tissues. Cod-liver oil would appear further to have a special power of aiding the regeneration and nutrition of the cells lining the respiratory tract, and

to be of great service in clearing up the intercurrent catarrhs which are so frequent in those prone to tubercle. To its value in chronic bronchitis we have already alluded (see p. 193).

The best time for taking cod-liver oil is soon after meals, when the stomach is occupied by food in a condition prepared for escape through the pylorus, for it is beyond the pylorus that the oil becomes absorbed; but some patients will in practice be found better able to assimilate the remedy at other times. In children, and sometimes in adults, the syrupus ferri phosphatis co. of the B.P.C., or steel wine are excellent vehicles, and various other agreeably flavoured tonics may be combined with the oil, such as quinine wine with a little hypophosphite of soda or lime, or phosphoric acid and strychnia with syrup of orange.

Certain additions may sometimes with advantage be made to cod-liver oil: thus, a drop of creosote or $\frac{1}{100}$ grain of strychnia (Williams) are valuable correctives. In a few cases it will also be found that the addition of an alkali by emulsifying the oil will enable it to be better borne and absorbed;* and there are now many excellent and comparatively palatable emulsions of cod-liver oil made in combination with malt extracts. Of these a simple emulsion of oil and malt extract in equal parts can usually be well borne, a dessert-spoonful or more two or three times a day being prescribed. The Emulsio Olei Morrhuæ Co. of the B.P.C. contains 50 per cent. of oil, and is an elegant preparation which may be recommended for persons of delicate digestion.

The most favourable periods for giving oil are the apyrexial intervals of phthisis. In small doses, however, the remedy can sometimes be taken with advantage, if not during the period of continued fever, at least during the more prolonged hectic of the disease.

If for any reason cod-liver oil cannot be taken, trial may be made of *Sodium Morrhuate*, a preparation lately introduced by Sir Leonard Rogers,[5] and containing the sodium salts of the unsaturated fatty acids of which cod-liver oil is composed. Small doses, commencing with ½ c.c. of the 3 per cent. solution, and increasing by 2-4 minims at each injection to 2 c.c., are given subcutaneously two or three times a week.

* Brompton formula: Mist. Olei Morrhuæ Preparata. ℞. Olei Morrhuæ ʒvi., Liq. Ammoniæ Fort. ♏ii., Olei Cassiæ ♏i., Syrupi ʒii. Dosis ʒii.

Intravenous injections, beginning with $\frac{1}{2}$ c.c., are then recommended, and should be cautiously increased. Should a febrile reaction occur the dose should be diminished. We have tried the preparation by subcutaneous injection and seen benefit result, but have not ourselves given it intravenously.

Creams and *fats* may be also prescribed. A piece of mutton suet allowed slowly to dissolve in a tumbler of milk, warmed by standing on the hob or in a slow oven, is an old-fashioned remedy for phthisis; the milk thus treated should be filtered through muslin before being taken. *Olive oil* taken alone, or with salads or sardines, is an imperfect substitute for cod-liver oil.

Of *general tonic remedies* in the early periods of phthisis, arsenic is one of the most valuable. The arseniate of iron or small doses of arsenious acid may be given in pilules with food twice a day. The late M. Jaccoud recommended $\frac{1}{70}$ grain of arsenious acid to be taken in pill form at the commencement of two principal meals, increasing the dose each week by two pilules, until six, eight, or ten are taken daily; this treatment being continued for two or three months unless signs of intolerance of the drug are shown. After this period the dose should gradually be diminished. This remedy is especially valuable in cases in which iron is not well borne. If under the arsenical treatment appetite fails, the tongue becomes coated, digestion painful, or the bowels relaxed, it must be at once restricted or withdrawn.

Unless specially indicated, strychnia and quinine are of no particular service at this period. The hypophosphites are of undoubted value, and also iron in small doses and for short courses.

Period of Active Softening and Formation of Cavities.—During this period the particular symptoms to be regarded are: (1) pyrexia; (2) nervous prostration and bodily exhaustion; (3) certain special symptoms—viz., wasting, anorexia, night-sweats, cough and expectoration, hæmoptysis and intercurrent pleurisies.

Pyrexia.—The fever at this period of phthisis, often hectic in type, is probably, as we have seen (p. 467), dependent in most cases upon the active growth of the tubercle bacillus, and the absorption of the tuberculous toxines so found. In a smaller proportion of cases true secondary infections are pre-

sent, and the organisms responsible take their share in the production of the pyrexia.

With regard to the treatment of such cases, we must remember that the growth of the tubercle bacillus and of the pyogenic and other organisms, with which it may be associated, is most favoured at the normal body temperature, and that the presence of fever may not improbably be regarded as to some extent a protective factor tending to inhibit activity and growth. It is probable also that phagocytosis is more active, and that protective bodies, such as antitoxins, bacteriolysins and agglutinins, are produced more readily when a moderate degree of fever is present than at the normal temperature of the body.[6] Whilst the growth of the bacillus and attendant organisms is most favoured by a normal temperature, the higher ranges of temperature, on the other hand, are associated with a negative phase, during which the phagocytic and other protective functions of the blood are depressed.

Our treatment of fever, therefore, must be cautious, and should be directed chiefly to heightening the resisting power of the patient by means of a generous diet and the stimulating effect of fresh air, thus enabling him to form additional protective substances and to check the growth of the tubercle bacillus and of other organisms which may be associating themselves in its activity. This effort on the part of the patient may be assisted by certain drugs, such as arsenic and iodine.

Arsenic itself cannot be given in these cases in sufficient doses to diminish the temperature in any striking degree, but it has a marked influence upon some of the most distressing symptoms attendant upon the fever; and it sometimes in a striking manner improves the general condition of the patient. This drug is most indicated in those cases in which daily recurring chills are complained of. Three to five drops of the liq. arsenicalis or of the arseniate of soda solution, or gr. $\frac{1}{12}$ to gr. $\frac{1}{4}$ of the arseniate of iron three times a day, will often suffice to prevent the recurrence of these chills. Arsenic must be given with, or immediately after, meals.

Another preparation of arsenic which we often prescribe, and which is less toxic and can therefore be administered in larger doses, is the cacodylate of soda (sodium dimethyl-

arseniate). This may be given by the mouth in doses of half a grain three times a day, or, better, by hypodermic injection, the patient receiving subcutaneously each day for fourteen days a single $\frac{3}{4}$-grain dose, dissolved in 1 c.c. (17 minims) of sterilised water. An interval of ten days should then be allowed before reverting to the injections. During the treatment due observation should be kept upon the knee-jerks and the fields of vision to insure that no adverse effects are being produced upon the nervous system, though we have never known such to occur. The cacodylate of soda appears to us to be more effective than simple arsenic, and when the fever has not been severe we have several times known it to abate, and the temperature to become normal after two or three courses of injections. In other cases, especially when there has been a syphilitic taint, we have obtained benefit from injections of neo-salvarsan.[7]

Should arsenical treatment fail, or the patient be averse to the injections, we should advise that trial be made of the *intensive iodine treatment*, introduced by Dr. David Curle.[8] This treatment consists in prescribing 20 grains of Potassium Iodide in half a pint of water after breakfast, and four hours later an ounce of Aqua Chlori in half a pint of freshly prepared and sweetened lemonade, thus effectually masking the nauseous taste of the chlorine. The dose of chlorine water is repeated at intervals of two hours until three doses have been given, and after two or three weeks a fourth dose may be added. The chlorine liberates iodine from the potassium iodide in the blood, and any beneficial effect is thought to be due to the antiseptic action of the nascent iodine thus produced. Should symptoms of iodism manifest themselves, 30 grains of Sodium Bicarbonate should be given every two hours, and, if the symptoms continue, the treatment should be stopped; but we have not often observed this complication.

We have tried this method on many occasions, and though we have never observed any sudden fall of temperature to follow its use, we have in not a few cases of moderate pyrexia observed a gradual lowering of the fever, and sometimes its complete subsidence, after some weeks of treatment.

During this hectic period *iodoform*, *creosote*, and *tar* have been recommended for internal administration on account of their antiseptic properties. We have ourselves found them

of more value in the apyrexial period (see p. 687). When there is much local disturbance of stomach and upper bowel, however, small doses of creosote, in combination with opium, are sometimes of considerable service.

Antipyretics must be employed with care, and should be prescribed with the idea of moderating fever when excessive, rather than of suppressing it altogether. It sometimes happens, however that the good effect upon the temperature persists after the discontinuance of the drug. The following antipyretic remedies may be considered:

(*a*) *Quinine.*—In from three to five grain doses, taken in milk and between meal-times, quinine is often of value in controlling hectic and in sustaining the patient. A tablespoonful of whisky may often with advantage be added.

(*b*) *Phenazone, antifebrin, phenacetin,* and allied drugs of well-known power in reducing temperature, are to be used in phthisis with great caution, and in carefully calculated doses prescribed at definite times with a view to moderating excessive peaks of temperature. In many cases they cause considerable nervous depression, with profuse sweating, and sometimes vomiting; in others they merely postpone the pyrexial rise to a later period of the day, and weaken the control of the nerve centres, thus exaggerating pyrexial fluctuations. In small doses, however, such as five grains of phenazone, or two to three grains of phenacetin, in combination with quinine, salicin or aspirin, these drugs are sometimes of assistance.

(*c*) *Cryogenin* (meta-benzamine-semicarbazide) is also of value in this direction. It is best given in cachet form in doses of seven or ten grains, about three hours before the maximum daily temperature is reached, and is often successful in producing a decided diminution of fever. No untoward symptoms seem to accompany its use, though, as with others of these drugs, its effect is too often temporary.[9]

The evening temperature can sometimes be sufficiently moderated without medication by the employment of tepid sponging with a solution of dilute acetic acid one part, water, at a temperature of about 85°, six parts, and eau-de-Cologne $\frac{1}{4}$ part. As an adjuvant to other remedies, especially when there are night-sweats, this application is of value.

In the comparatively rare cases in which a true secondary infection is present (see p. 467), if the fever continues in spite of treatment, a cautious attempt may be made to check its progress by means of an appropriate vaccine. We have not, however, observed much benefit from this line of treatment.

Acute cases of the kind which we are considering, provided the disease is restricted to one lung, are sometimes greatly benefited by the induction of an *artificial pneumothorax*, a method of treatment which we shall consider in a later chapter (p. 719). This form of treatment should always, therefore, be borne in mind, and must be carefully considered, as soon as it has been shown that the pyrexia is not yielding to more ordinary methods of treatment.

Cough: its Local and General Treatment.—Except for cases in which throat symptoms are prominent, sprays are of little value, since it is very doubtful whether they penetrate beyond the larynx or main bronchi.

There is no doubt, however, that by the inhalation of the dry vapours of volatile antiseptics much advantage may be gained in this stage of the disease. The effect of such inhalations is to lessen expectoration and to relieve cough; and one of their chief functions is to diminish the necessity for cough mixtures.[10] Certainly the cough linctus treatment of this eliminative period of phthisis, by continually lulling cough and deranging stomach, is the very worst that could be devised. Our great objects are to get rid of the effete products of caseous liquefaction and suppuration, and to keep the pus-secreting surface as disinfected as possible without harming the patient in the process. The cavity contents are, it is to be remembered, in contact with living tissue, which has a retarding influence upon bacterial activity; and it is from this point of view that we look to general tonic and hygienic measures as operative in aid of our more special medication.

The majority of patients find close naso-oral respirators irksome, but can wear the more open and lighter pattern devised by the late Dr. Burney Yeo for a longer time and with less fatigue. The pattern long in use at the Brompton Hospital is of this type, and consists of perforated zinc, bound round the edge where in contact with the face with chamois leather. A small receptacle is provided for the cotton-wool, which must be moistened with the solution for inhalation.

This respirator is inexpensive, and may be obtained from Messrs. Maw and Sons. Other forms are those of Coghill and Roberts.

A good inhalant consists of a mixture of the following: Tincture of Iodine ʒi., Liquefied Carbolic Acid ℥ii., Creosote ℥ii., Spirit of Ether ʒi., and Spirit of Chloroform ʒii. We have also found three drachms of eucalyptol or olei pini sylvestris to the ounce of rectified spirit, or spirit of chloroform, a good combination: twenty drops on the wool of the respirator, to be used for half an hour to an hour or longer, after the first morning expectoration, in the middle day and in the evening. If the cough be troublesome, one drachm of oil of bitter almonds may be added to the one-ounce solution, and double strength spirit of chloroform used as the solvent. If the patient be able by assuming a dependent posture and by a determined effort more completely to clear the cavities before the use of the respirator, it is well to do so. This must be decided at the discretion of the doctor. In some cases the respirator may be worn almost constantly, such "continuous inhalation" of antiseptic vapour being thought by the late Dr. Lees to exercise a direct inhibitory effect upon the activity of the tuberculous process in the lung. We have ourselves never seen such inhalation produce in febrile cases any marked alteration in the temperature chart, and believe that the good effect is restricted to a lessening of the bronchial catarrh and its attendant cough.

Another valuable prescription from the Brompton Hospital Pharmacopœia is the following: Menthol ʒii., Creosote ʒi.ss., Spiritûs Camphoræ ʒi.ss., Spiritum Rectificatum ad ℥i. In some cases, especially when the cough is frequent and expectoration abundant, a large bib may usefully be employed, sprinkled with a weak formalin solution ($2\frac{1}{2}$ to 5 per cent.). This has the double advantage of serving as an antiseptic inhalation, weak but constant, and of catching the spray of the cough in bedridden patients.

A good sedative inhalation, especially when the larynx is involved, is produced by placing a few crystals of menthol in a dry tumbler and immersing it in hot water. In cases also where there is much tracheal irritation, causing incessant cough, ten or twenty drops of chloroform, inhaled from a smelling-bottle or a handkerchief stretched across the open

mouth, will give great relief; but this remedy must be used with caution and watchfulness. It must be remembered that cough is the only means by which the phthisical lesions can be drained of their deleterious products, and cough cannot therefore be smothered by sedative remedies without damage to the patient.

There are certain times and kinds of coughing which call for special treatment, particularly (*a*) the irritable and ineffectual cough, (*b*) the night cough, (*c*) the early-morning cough.

(*a*) *The irritable and ineffectual cough,* in which only a very scanty expectoration follows after prolonged and exhausting efforts, can to some extent be controlled by the patient himself, and he should be encouraged to repress it as far as possible. In other cases treatment is indicated, and the symptom may be best modified by the use of one or other of the sedative respirators above mentioned. A small dose of opiate, such as five minims of nepenthe, or heroin gr. $\frac{1}{12}$, may for a time be added to the day mixture. These irritable coughs generally mean that caseating and softening lesions have not yet established communication with the bronchi, and with this occurrence the cough becomes looser and more easy.

(*b*) *Night cough* requires sedative treatment by opium, chloral, heroin or codein. A combination of small doses of these drugs, made up to a tablespoonful with mucilage and chloroform water (see p. 672), is often valuable, and may be repeated in half-doses once or twice in the night if necessary. The Pilula Ipecacuanhæ cum Scillâ of the Pharmacopœia is also useful. When the cough is described as very "tight," an opiate may be taken in an alkaline effervescing draught at bedtime.

(*c*) *The morning cough,* to which we have already alluded (p. 673), requires quite a different treatment; it should never be checked by sedatives. Its severity and the amount of expectoration are generally in inverse proportion to the quietude of the night. Expectoration and clearance of cavities should be encouraged by hot stimulating drinks, such as a cup of hot coffee or milk (with perhaps a liqueur glass of cognac or rum), or a dose of ether and ammonia. Sometimes a hot, stimulating poultice over the sternum is useful; for example, a linseed poultice, with powdered camphor, in proportion of one ounce camphor to one pound linseed. It

is an advantage if the lungs can thus be cleared before breakfast, as there is then less tendency for the cough to induce vomiting. We may here allude to the highly irritating effects upon the stomach of the products of caseous softening and cavity secretion, when swallowed. Some patients, delicate-minded females and sensitive men, feel a reluctance or a timidness in expelling the products of their disease; children especially fail to do so. This inability or unwillingness to eject the sputa is a fruitful source of stomach and bowel troubles, and of local tuberculosis; a simple warning will often prevent the necessity for special medication. A pleasant antiseptic mouth-wash should be used after the morning expectoration.

Night-sweating.—This is a symptom which usually calls for treatment from time to time during the eliminative period of phthisis, but is of much less frequent occurrence since the introduction of the open-air lines of treatment. It arises from two principal causes—viz., fever and nervous exhaustion, these causes being commonly combined, but the one or the other predominating in different cases.

There are innumerable empiric remedies for night-sweats, all of them successful in certain cases. They should not, however, be used until such rational measures, as are indicated by the causative conditions above alluded to, have been adopted.

The patient must be steadily supported by nourishment during the day, and some readily digestible food, such as strong beef essence, given the last thing at night. Fever must be moderated by tepid acid sponging at bedtime (a wineglassful of aromatic vinegar with eau-de-Cologne to a tumbler of water), and a third dose of the quinine, or acid tonic in use may be given a little before bedtime. In many cases we have found a mixture containing two grains of quinine sulphate and twenty minims of dilute hydrobromic acid of value. If these means prove insufficient, then aid may be sought from specific remedies. Extract of belladonna gr. $\frac{1}{2}$, or atropine gr. $\frac{1}{100}$, may be given in the form of pill at night either alone or in combination with quinine. If the cough is troublesome, hyoscyamine gr. $\frac{1}{100}$ may be preferred. When the hectic has lasted some time, and has resulted in much nervous exhaustion, strychnia is useful in full doses at bedtime, but this remedy is sometimes unfortunate in causing wakefulness.

Four-grain doses of oxide of zinc, or two grains of valerianate of zinc will sometimes answer alone or in combination with belladonna.

In cases in which night-sweating is observed to be preceded by shallow breathing and slight livid pallor, strychnine is especially indicated. A current of oxygen occasionally brought near the patient during sleep may also prove of service.

Agaricin gr. $\frac{1}{12}$, and camphoric acid gr. xv., are other remedies of occasional value, their good effect being apparently due to their depressing action upon the nerve-endings to the sweat-glands.[11]

It is of the utmost importance to have some easily assimilable food, such as good cold beef-tea or beef-essence, ready to hand, should the patient wake up, and especially with night perspiration, for the sweats are profoundly depressing from the large quantity of saline material discharged, and the nervous exhaustion thus induced tends to perpetuate their nightly recurrence. The immediate supply of a stimulating salt-containing food tends to remedy the loss and to prevent its recurrence. Patients liable to night-sweating should wear a thin flannel loosely-fitting over-gown, and a fresh night-dress should always be ready aired for changing.

General Summary.—Throughout the variable but often prolonged period of suppurative fever through which the patient has to pass during the softening and elimination of the caseous products in the more active forms and periods of phthisis, the lines of treatment to be followed may thus be summarised.

(*a*) The patient must be kept absolutely at rest in bed, and open-air lines of treatment strictly enforced. If the conditions are satisfactory, this can be well carried out in the patient's own home, a shelter being erected in the garden, in which he may lie out, and, even under favourable conditions, spend the night. Treatment in a sanatorium in this stage of the disease is not essential, and, indeed, cases of this kind are not altogether welcome in such institutions, requiring, as they do, additional attention and nursing. In cases where the home is situated in the midst of damp and unhealthy surroundings, a removal to a high and dry locality, or to the seaside, with sunny exposure, cannot fail, *ceteris paribus*, to

be beneficial. No climatic change of any radical kind is indicated for this period of the disease. For the poorer classes our large special hospitals are admirably adapted. Unfortunately febrile cases of the type considered in this chapter are not always welcome even here, but it is clearly the duty of every such hospital to provide for a certain percentage of such patients. In every county there should also be told off a certain portion of the infirmary, so reconstructed as to afford especial comfort and good hygienic conditions, and thus adapted for active as well as advanced cases of phthisis.

(*b*) The patient must be steadily supported by an abundant and well-assorted dietary, rich in fats and nitrogenous elements. If milk can be borne, one or two pints should be taken daily, as the patient is usually somewhat wasted. In some cases a light wine or beer, or other form of alcohol, is of value.

(*c*) Particular symptoms—pyrexia, dyspepsia, sweatings, cough, and expectoration—will suggest the line of medicinal treatment, which must be determined upon after careful consideration of the whole case, and not lightly changed. A written sketch of the dietary and times of taking medicine will be a useful guide to friends and nurses, and a wholesome check against mixtures "every four hours" and cough linctus "occasionally," besides pills for night-sweats, and local applications, a wholesale medication, which if persisted in cannot fail to be disastrous.

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CHAPTER XLVIII

TREATMENT OF PULMONARY TUBERCULOSIS—(*Continued*)

The more Quiescent Period—Contracting, Secreting, and Ulcerous Cavities—Fibroid Stage.

In dealing with the pathology of pulmonary tuberculosis, we pointed out how the disease, in most of its varied manifestations, shows a disposition to periods of quiescence; and it is during these periods, sometimes occurring in the earlier stages, sometimes after a prolonged hectic stage resulting in completed excavation of a large portion of a lung, that an appropriate and definite treatment may succeed in producing permanent arrest. It is at these periods also that most may be expected from climatic change (see p. 636).

In these stages the preparations of creosote and its congeners, especially guaiacol, are of distinct value. The use of these remedies, long since suggested and abandoned, was resuscitated when the specific nature of tubercle was finally demonstrated. Bouchard and Gimbert in France,[1] and Sommerbrodt[2] in Germany, appear to have been the first to reintroduce them, and they were long in use by the French physicians before they became seriously employed in this country. Their value in many cases of phthisis, and especially at the stages indicated, cannot be doubted, although the exact mode by which their effect is produced remains obscure.

Creosote and guaiacol may be prescribed in the form of capsules, containing from one to three or more minims. A two-minim capsule should be taken, in the first instance, immediately after food three times a day, and the dose gradually increased. Some patients can take the drugs better when held in solution by rectified spirit, and flavoured by a liqueur or bitter. Cod-liver oil is another good vehicle; doses up to 10, or even 20 minims of the creosote being dissolved in two

drachms of the oil, to be taken twice a day after food. Most patients are able in this way to tolerate the required dose of the drug without derangement of the stomach. It is not as a rule necessary to prescribe very large doses of the drug, but moderate doses should be persisted with for a long time. Other formulæ which we have found of practical value have been the following:

(*a*) ℞ Guaiacol carbonatis, Guaiacol benzoatis vel Styracol ʒi.ss.
Calcii hypophosphitis ... ... ʒss.
Pulvis Tragacanthæ Co. ... ... ʒi.

Misce bene, adde guttatim :

Syr. Pruni Virginianæ vel Elixir Aurantii (U.S.P.)... ℥ss.
Syr. Calcii Lacto-phosphatis vel Syr. Hypophosphitum Co. ... ... ℥i.
Aquam Chloroformi ... ... ad ℥vi.

One tablespoonful in water or liquid malt three times a day soon after food.

(*b*) ℞ Creosoti Carbonatis ... ... ʒiv.
Tinct. Gentianæ Co. ... ... ʒiv.
Syr. Pruni Virginianæ ... ... ad ℥iii.

One teaspoonful in a wineglass of water or malt extract after food three times a day. Increase the dose by five drops each second day up to two teaspoonfuls by measure.

It is sometimes desirable, in patients with delicate digestion, to secure that the creosote preparation shall pass through the stomach undissolved, to be absorbed from the intestinal tract. This may be effected by the use of "proposote," a combination of creosote and phenyl-propionic acid introduced by Messrs. Parke Davis and Co. Proposote is insoluble in water and acids, but is decomposed by alkaline fluids into creosote and phenyl-propionic acid. It is best prescribed in capsules, 10 minims corresponding to 5 minims of creosote.

The idea of destroying the tubercle bacillus by any internally administered antiseptic has been abandoned, and the effects of creosote treatment, when successful, are recognised as chiefly to diminish expectoration, to lessen waste of tissues, and to render them less susceptible to tuberculous activity.

Quiescent, Secreting, and Ulcerous Cavities.—We may now pass to a consideration of the cavities, which form so conspicuous a feature in many cases of phthisis. In certain patients, after the pulmonary disease has ceased to extend,

the cavities, varying in size and number, become quiescent, and slowly contract, yielding less and less secretion. In these cases of drying and contracting cavity the cough becomes irritable, and the patient frequently complains of its "tightness," having been accustomed to easy expectoration whilst the secretion was abundant. He should be encouraged by explaining to him the favourable nature of the cause of his difficulty, and directed to check, so far as possible, by an effort of will, the tendency to violent cough. Sedative cough mixtures, or the addition of $\frac{1}{12}$ gr. of heroin to the day mixture, etc., injurious whilst the expectoration was abundant, now, judiciously timed, become of much value. More or less morning expectoration persists for a considerable time after the cough has ceased during the day, and should not be checked by sedatives. It must not be forgotten, too, that the morning expectoration may for months, and even for years, contain a certain number of more or less ill-conditioned tubercle bacilli which do not necessarily affect the patient's general well-being, since they are shut off by the dense fibroid surroundings of the cavity. The further hygienic, climatic, and medicinal treatment of cases of this kind must be conducted on general principles.

It is of great importance to keep such patients in a pure atmosphere, since they are most sensitive to all septic conditions. Under unfavourable hygienic circumstances the cavities become secreting, and other surrounding centres of disease take on fresh activity.

Moorland, sea, and mountain air are all suitable for these cases, and one or other should be advised in accordance with the special indications (see chapters on Climatic Change). As the patient's strength improves, the development of the sound portions of the lungs may be encouraged by carefully regulated exercise on rising ground. It is in this latter phase, provided the *extent* of lesion be not too great, that mountain resorts prove so valuable.

Secreting Cavities may, as already pointed out, persist from the acute stage, or be developed by renewed activity of the lining of cavities which had become quiescent.

The objects which we have in view in the treatment of such cases, are—(1) to lessen secretion; (2) to promote its evacuation; and (3) to disinfect the cavities.

Counter-irritation is useful over the cavities in the form of strong iodine applications, or flying blisters, or perhaps a blister kept open for a time by savin ointment. A course of mineral acids, in combination with bark or astringent iron tonics, with cod-liver oil, is from time to time needed.

It is in these cases in which, with little or no pyrexia, there is yet considerable secretion from cavities, that the preparations of tar, creosote, or eucalyptus may often be given in moderate doses with great advantage. Tar may be best prescribed in a morning and evening dose of the *eau de goudron* of Guyot, from ʒss. to ʒi.ss. in a wineglass of plain or some mineral water (Ems, Bourboule, Vichy), to which a little warm milk, and, if necessary, cognac may be added; a single, or at most two, doses of tonic being given during the day. The tonic may from time to time be varied or omitted, but the tar preparation should be persisted in for weeks or months. Bell's Liquor Picis Aromaticus, in doses of twenty to forty minims (2 to 5 grains of tar), is also a useful and not unpleasant preparation ; or the tar may be prescribed simply as a pill.

In cases, especially, of intercurrent catarrh, causing an increased secretion from quiescent cavities, five or ten drops of a mixture of equal parts of oil of eucalyptus and pure terebene, taken on sugar three times a day between meals, may be prescribed with advantage. When the secretion is very abundant and the condition of the patient depressed, we have found oil of eucalyptus, held in solution with a drachm of rectified spirit, with the addition of glycerine and a little dilute phosphoric acid and quinine, a useful combination.

Subcutaneous, or rather intramuscular injections of creosote or guaiacol, dissolved in sterilized oil, have been employed in this, and also in the more active stage of phthisis. We have had opportunities of noticing the results of this method of treatment, but have been by no means convinced of its superiority; the imagination of the patient cannot fail for a time to be impressed, but at the cost of considerable discomfort, and in some instances of no little positive suffering, local necrosis sometimes occurring at the seat of injection.

If the opsonic index with regard to any particular organism in the sputum (whether streptococcus or other) is found to be

depressed, it may be raised by the employment of a suitable vaccine, under which treatment the amount of secretion will sometimes diminish.

Sedative cough mixtures are directly contra-indicated except at bedtime, and then solely for the purpose of procuring rest.

Seaside resorts, the more bracing of the Riviera stations, are the climates most suitable for these cases in the first place, but many will after a time continue to make better progress in the dry Alpine or South African highlands, provided the other indications for such climates are fulfilled (see Chapter XLV.).

Ulcerous Cavities.—Cases of ulcerous or active cavities are always to be regarded as probably due to insanitary surroundings, and such evil conditions must be first looked to and remedied. These cases are of an erysipelatous type, and under the more recent methods of hygienic treatment are now rarely met with; they are best treated by quinine in full doses, or perhaps tincture of iron, and locally by sedative inhalations of carbolic acid, conium, and chloroform, with hot-water vapour.*

After the more active general symptoms have lessened, if the blood-stained and copious expectoration lead us still to infer that the walls of the cavity are hyperæmic, if not ulcerated, the best treatment will be found to be free counter-irritation. A blister should be applied over the site of the cavity, and should be kept freely discharging for a week or ten days by means of savin ointment dressing. We have seen the active local symptoms completely subside under this treatment, and the expectoration, from being abundant and sanguineous, become scanty and viscid, apparently consisting of bronchial mucus only.

With regard to the possibility of surgical intervention in the treatment of phthisical cavities, we must refer the reader to a later chapter (p. 728).

Chronic and Fibroid Stage.—The management of the chronic and fibroid stages of pulmonary tuberculosis consists chiefly in the prevention of fresh catarrhs, which may lead in turn to the further spread of disease. To this end all insanitary conditions must be avoided, clothing should be suitable,

* Chloroformi ♏x., Succi Conii ʒi., Glycerinum Acidi Carbolici ad ʒii., Aq. Bullientis ℥viii.—*Brompton Hospital Pharmacopœia.*

and the diet nutritious but not stimulating. When practicable, also a more genial climate should be selected, thus insuring protection from irritating fogs and cold damp winds. Iodine applications, soothing or antiseptic inhalations (carbolic acid being particularly useful when there is any fœtor of expectoration), seem the best local remedies. The general condition, including that of digestion, and the nature of the cough and amount of expectoration, supply us with indications for the administration of appropriate drugs—iron, cod-liver oil, strychnia, alkalies, tonics, creosote, etc.—or warrant the withdrawal of all medicines.

There is only one special remark concerning the treatment of these chronic indurative cases of phthisis, during the often extended period of quiescence, which seems called for, and it is this: that, having lived to acquire their immunity, such cases do not require the persistent administration of tonic medicines and cod-liver oil, although needing careful *surveillance*, and for several years, where practicable, carefully selected climates to suit the different seasons of the year. They improve immensely under such remedies up to a certain point, which may be readily recognised by the medical attendant, and cannot be better described than by saying that it amounts to the most perfect health attainable by a patient who has had a certain area of respiratory surface cut off. If beyond this point we persevere with iron and oil, and too nourishing or stimulating a diet, we may still further increase weight and heighten colour, but the pulse quickens, the patient gets more short of breath, he becomes, in a word, plethoric, and liable to pulmonary congestion and hæmoptysis, or to dyspepsia and diarrhœa. A rapid neutralisation of all the good results obtained, with great danger of fresh, and perhaps fatal renewal of the old disease, is thus the consequence of too great anxiety to arrive again at a degree of health and bodily vigour which is impossible with a permanently damaged lung.

The treatment of pulmonary tuberculosis occurring in the course of *diabetes* calls for no detailed comments. In such cases a considerable abatement in the régime for diabetes is usually necessary, and codeia, quinine, and cod-liver oil are indicated.

REFERENCES.

[1] "Note sur l'Emploi de la Créosote vraie dans le Traitement de la Phthisie Pulmonaire," par MM. Ch. Bouchard (de Bicêtre) et Gimbert (de Cannes), *Gazette Hebdomadaire de Médecine et de Chirurgie,* 1877, No. 31, p. 486.

[2] "Ueber die Behandlung der Lungentuberkulose mit Kreosot," von Professor Dr. Julius Sommerbrodt (in Breslau), *Berliner Klinische Wochen schrift,* 1887, No. 15, p. 258.

CHAPTER XLIX

TREATMENT OF THE COMPLICATIONS OF PULMONARY TUBERCULOSIS

Ulceration of the Bowel—Laryngeal Tuberculosis—Hæmoptysis—Vomiting—Intercurrent Pleurisy—Meningitis.

Ulceration of the Bowel.—In the treatment of ulceration of the bowel in phthisis it may be usefully remembered that the mere presence of such ulcers does not necessarily cause diarrhœa, this symptom, when present, being due to increased peristalsis set up by an active condition of the ulcer, or to attendant catarrhal inflammation of the adjacent mucous membrane. We have thus two conditions to bear in mind in the treatment of tuberculous ulceration of the bowels—viz.: (1) acute ulceration, the first stage of the disease, in which the ulcers are in process of formation or activity; (2) chronic ulceration, associated with diarrhœa alternating with constipation, or with attacks of intercurrent looseness of bowel arising from errors of diet.

Acute Ulceration.—In this condition, the symptoms of which have been already described (see p. 542), the diet must be carefully regulated, so as to give as little residue as possible. The patient must be kept warm in bed, and food restricted to milk and a little good beef-tea or meat essence. Four or five ounces of milk, citrated if necessary, should be allowed every two hours, and at certain intervals the milk may be replaced by beef-tea, half a pint of which, equivalent to half a pound of meat (see p. 330), may be allowed in the twenty-four hours. The milk can be given warm or cold, in accordance with the desire of the patient; sometimes a little crushed ice may be added, or it may be boiled with rice and strained, when six ounces should be allowed instead of four. If further nutriment is required, the milk should be

thickened three or four times a day with a little plasmon or sanatogen. In other cases Benger's, or other pancreatised or malted farinaceous food, may be prepared with the milk twice or thrice daily. Sometimes koumiss in the third degree of fermentation (see p. 603) may be substituted for milk. The beef-tea can be varied with veal-tea, chicken or mutton broth, and the latter may occasionally with advantage be added to the milk. After a few days, rusks and a little tea may be allowed, and the diet slowly improved through such grades as custards, ground-rice puddings, invalid turtle, scalded bread-and-milk, pounded chicken, and the like. As the diet is improved, the intervals between the times of taking food should be increased to three or four hours.

In some cases a small quantity of stimulant in the form of brandy, added to each second quantity of the milk, is of service, or a little old port wine may be allowed.

During the acute period now under consideration, and whilst any tenderness remains, the patient will be best in bed, and a pine wool pad, or calorigen or thermogen wool, should cover the abdomen. When there is marked tenderness over the region of the cæcum, a small blister may with advantage be applied in this situation under a poultice.

In medicinal treatment it is of importance, first of all, to clear away any irritating matter from the bowel, and the administration of from one to two drachms of castor oil, well shaken up with two ounces of hot milk, and a dessert-spoonful of cognac added, will prove of service. It is often a good plan to give a small dose, half to one grain, of calomel two hours previous to the dose of oil. After the lapse of a few hours, to allow the effect of these medicines to take place, ten or fifteen grain doses of bismuth, with half a grain of ipecacuanha, and ten grains of sulphocarbolate of soda in mucilage and chloroform water, is a good routine treatment. After some eight or twelve doses, a little chalk mixture should, if needed, be added in place of the soda. Sometimes cerium will answer better than bismuth, and in cases of painful peristalsis small doses of opium are needed.

When there is much tympanites and tenderness, five minims of glycerine of carbolic acid, thirty minims of the solution of perchloride of mercury, and five minims of tincture of opium, with compound tincture of chloroform, a little sul-

phocarbolate of soda, and some aromatic water every four hours for a day or two, may be of great service. In some of these cases carbonate of guaiacol will be found useful, especially after the more acute stage has passed.

It is the greatest possible mistake to treat tuberculous diarrhœa with astringents and opiates as a matter of routine, and without very carefully looking to the diet. This treatment is on a par with that of pulmonary lesions by sedative cough mixtures, and only serves to mask symptoms whilst the tuberculous lesions are spreading and deepening.

Chronic Ulceration.—In this condition, if diarrhœa supervene upon previous constipation, or if the appearance of the motions and coated tongue lead to the inference that scybalous or irritating materials are present in the bowels, a dose of castor oil or compound rhubarb powder should first be given, the dietary carefully gone over although with less strictness than in the acute stage, and a course of bismuth and soda or chalk prescribed.

In severe cases twenty grains of bismuth and five grains of Dover's powder is a useful combination, or ten grains each of bismuth and compound kino powder may be given every three or four hours. In a much more limited number of cases sulphuric acid and opium answer better. When there is much tenderness and tympanites, a combination of carbolic acid, mercury, and opium, as above stated, may be given for a day or two. A layer of pine wool quilted on flannel should be kept firmly applied to the abdomen. On the subsidence of active symptoms a little calumba, cascarilla, or quinine, may be given twice a day in combination with fluid bismuth; or five-grain doses of hypophosphite of lime may be prescribed with ten minims of Liquor Calcis Saccharatus in calumba or chiretta infusion.

Constipation is very apt to follow upon diarrhœa in cases of ulcerative disease, and a collection of solid fæces tends to reawaken the activity of the ulcers. The smallest available dose of laxative should therefore be prescribed when necessary, one drachm of castor-oil being frequently sufficient when taken well emulsified in hot milk. The diet may be modified, and a little fruit, cider or light beer allowed to correct the tendency to constipation, care being taken to exclude fibres, pips, seeds and skins of fruits and vegetables. In cases where

the third-stage koumiss has been taken during the period of diarrhœa, a return to medium koumiss may be suggested as less astringent.

The above plan of treatment will be found most generally efficacious in cases of moderate intensity of acute and of chronic intestinal ulceration, and during periods of intercurrent diarrhœa. In certain cases of chronic ulceration with diarrhœa, which have not yielded to ordinary methods of treatment, although the general health is fairly maintained, encouraging results have been obtained with heliotherapy (see p. 641). Cases of tuberculous peritonitis have also been thus influenced favourably.[1a]

In advanced cases the diarrhœa must, as far as possible, be restrained by careful diet, but our therapeutical resources are often taxed to the utmost, and in vain, to effect more than a passing relief. A remedy which scarcely ever fails to give temporary relief, even in the worst cases, is the starch and opium enema. Some acetate of lead may be added to this enema, or the lead and opium suppository may be employed with advantage. In more chronic cases and those of great severity we still have a large armoury of more decided astringents to fall back upon, amongst which acetate of lead and sulphate of copper (gr. $\frac{1}{4}$), with opium, hold a high place. But the vegetable astringents—kino, catechu, hæmatoxylin, tannic acid, Indian bael—may be each in turn tried in combination with opium, with decided, but often only temporary, benefit. The aromatic chalk and opium powder of the Pharmacopœia given in a mixture containing tincture of catechu, is a favourite remedy in these cases. Although all the vegetable astringents owe their efficacy to the tannin they contain, yet there is some peculiarity in each, and when one has failed another will often succeed, again in its turn to lose its virtues in the particular case. Almost all these cases require opium in addition to the astringent, and sometimes opium alone given in the solid form is the best remedy.

Laryngeal Tuberculosis.—The treatment of tuberculosis of larynx, although in many cases unsatisfactory, is by no means so gloomy a procedure as is sometimes imagined. The cardinal principle to be observed is to give rest to the larynx by checking cough and enjoining silence, keeping the patient meanwhile under the best hygienic conditions possible. Pro-

vided the pulmonary lesion be of favourable type and amenable to treatment, arrest of the laryngeal disease not infrequently occurs, and we have known not a few cases of laryngeal tuberculosis, some of which had proceded to ulceration, thus undergo arrest. We may now sketch out the treatment in greater detail.

If the laryngeal trouble shows itself when the pulmonary disease is in a comparatively early stage, and this is by no means uncommon, treatment in a sanatorium should be advised, since in addition to such local measures as may be required, the patient will have also the benefit of the general régime. As we have pointed out in a former chapter, the sanatorium chosen should not in this instance be at too high an altitude, or in too cold and bracing a locality. An attempt must also be made to insure as complete rest as possible to the parts by urging the patient to *refrain from all unnecessary coughing.* An irritable cough may often be allayed by the use of an antiseptic inhalation, containing menthol and creosote, or other remedies, as we have indicated in an earlier chapter (p. 681), and we must refer the reader to what we have there said in regard to the treatment of this symptom. Measures to check unnecessary coughing must be supplemented by enjoining a period of *absolute silence.* In some cases this may be enforced for as long as five or six months, but its duration must depend a good deal upon the temperament of the patient. Occasionally it leads to considerable mental depression, and must then be shortened. Such patients can, of course, converse by writing, and any monotony and depression may be lessened by reading cheerful literature.

Should the laryngeal affection be unaccompanied by much infiltration or œdema, the patient can be instructed in the method of intralaryngeal heliotherapy, directing the sun's rays on to the larynx by means of a laryngeal mirror held in position by himself.[2] If there be much infiltration of the parts, the method is contra-indicated, since reactive swelling will follow the treatment and might lead to dangerous dyspnœa.[1b]

In these cases, as we have already mentioned, the internal administration of creosote or guaiacol is often helpful.

In regard to direct treatment, too great a diligence in the use of local applications to the larynx is to be deprecated, and

as a general principle topical applications, except in the form of soothing sprays or inhalations, are better avoided. A simple remedy which often gives relief, by removing irritating secretion from the upper region of the larynx, is a spray of Ems water, slightly warmed by standing in a tumbler of hot water. In certain cases, however, when there is persistent swelling and infiltration of the larynx, which does not yield to the simpler methods of treatment which we have sketched out, puncture with the galvano-cautery in one or more situations may be sanctioned, with a view to induce sclerosis of the adjoining parts and thus imitate Nature's method of healing. We have seen benefit from this procedure, and carefully carried out, have not known it lead to harm.

The free curetting of ulcerated surfaces sometimes practised is not a method of treatment which we can recommend; we have known rapid spread of the pulmonary disease and death to follow such a procedure. The plan also of treating ulcers of the larynx by the direct application of lactic acid, 50-75 per cent., formerly so much employed, has of late fallen out of favour as not being in harmony with modern methods of treatment.

If the ulceration be extensive and diffused, palliative treatment is as a rule alone possible. When there is much pain the persistent use of external counter-irritation will be found most beneficial, especially when combined with that complete rest to the larynx which pain always calls for. A small blister, the size of a shilling, should be daily applied over the region of the larynx for several days, so as to keep up constant, but not too severe, counter-irritation.

Perhaps, however, the most distressing symptom which attends advanced tuberculous laryngitis is dysphagia, which so often follows ulceration of the epiglottis. This may be relieved by the application of a 10 per cent. solution of cocaine to the throat, or by allowing gelatine lozenges, containing 5 per cent. of the drug slowly to dissolve in the mouth shortly before meals. Burroughs Wellcome's or Oppenheimer's atomiser, charged with parolein and cocaine (5 per cent.), with a little morphine or chloral, will also afford great relief in many cases. In others an insufflation, composed of iodoform and boric acid (gr. 1 of each), and morphine acetate (gr. $\frac{1}{6}$), may be recommended.

Orthoform is another preparation which we, in common with other observers, have found very valuable in these cases. It is a synthetic product analogous to cocaine, but without its toxic defects; it relieves the pain very decidedly in many cases, and seems to produce no injurious results. The remedy may therefore be entrusted to the patients themselves, who should be directed to place a little of the powder on the palm of the hand, then cover it with the broadened end of a Leduc's auto-insufflator* (Fig. 65), the other end of which has been previously placed in the mouth and hooked over the back of the tongue; a deep breath is then taken, when the inhaled air draws the powder through the tube, and distributes it over the surface of the larynx.

If there be much inflammatory secretion over the ulcerated surfaces, resorcin may be added to the orthoform in the proportion of resorcin one part, orthoform three parts. If the

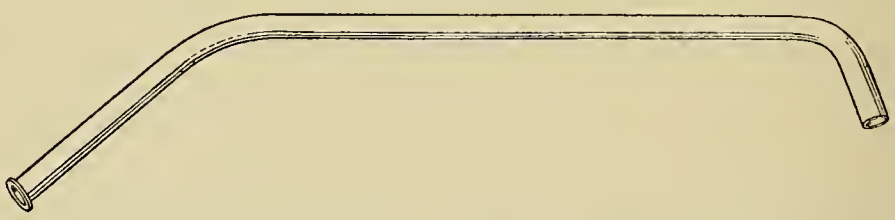

FIG. 65.—A LEDUC'S GLASS TUBE FOR THE INHALATION OF POWDERS INTO THE LARYNX (REDUCED TO HALF-SIZE).

dysphagia results merely from swelling of the larynx, without ulceration or abrasion of the surface, orthoform is of less value. In such cases insufflations of another synthetic product of a similar order, *anæsthesin*, used in the same manner as orthoform, will often prove successful.

When the pain in swallowing is severe, an injection of alcohol (about 1 c.c. of a solution consisting of 2 grains of hydrochloride of eucaine B. in an ounce of 80 per cent. alcohol[3]) into one or both superior laryngeal nerves often gives striking relief. This results from the partial anæsthesia of the larynx, which is brought about, and which lasts for a considerable time.

Patients in this stage sometimes swallow their food with greater ease when lying on one side or on the back; others again swallow best when lying with the head over the side of the bed, and sucking the liquid through a tube from a cup

* These simple and inexpensive glass tubes, which the patients soon learn to use with ease, may be obtained from Messrs. Maw and Sons.

placed at a lower level. In other instances nutrient enemata should supplement for a time the ordinary method of feeding; they help to give rest to the diseased parts, and lessen the distressing sense of exhaustion from which the patients often suffer.

When extensive ulcerative destruction of the vocal cords is present, the most distressing symptoms arise from the strain of cough and the mechanical difficulty of expectorating. These truly terrible symptoms are most difficult to relieve. When they arise a few drops of chloroform, or thirty drops of equal parts of chloroform and menthol, may be placed on a handkerchief or sprinkled on blotting-paper in a wide-mouthed smelling-bottle or tumbler, and inhaled. Finally, recourse must be had to morphia to lessen the acuteness of the distress.

Tracheotomy is very rarely necessary in cases of tuberculous laryngitis. Occasionally, however, the swelling of the parts becomes so extreme, perhaps from superadded septic inflammation, that it cannot be avoided. The effect of tracheotomy is to render expectoration almost impossible, and hastens rather than hinders the progress of the case.

Aphthous Mouth and Throat is a distressing complication of advanced phthisis, and one especially apt to occur in cases associated with diarrhœa. It may be warded off for a long time by careful cleansing of the mouth each time food is taken with weak boracic lotion, or with tepid water, just made pink with toilet Condy, to which a few drops of eau-de-Cologne have been added. A weak glycerine of thymol lotion is also useful.

Pregnancy and Lactation.—We may perhaps consider at this point the effect which pregnancy has upon tuberculosis of the lungs. There can be little doubt that this complication has not by any means that uniformly bad effect upon the pulmonary disease with which it has been sometimes credited.

During the pregnancy the general health is not uncommonly improved, and we have known patients, the subject of quiescent disease, go through more than one confinement without any harmful result. More than ordinary care must, of course, be taken in regard to rest and diet during the period of gestation, and the confinement should not be allowed to be unduly prolonged. In such cases we should not interfere in any way with the pregnancy.

It is later, with lactation, that the danger arises, the drain on the mother's system lowering her resistance and tending to light into activity the pulmonary disease. It is our practice, therefore, to forbid the suckling of the child by the phthisical mother except perhaps for the first few days.

It is well also to bear in mind that though the danger of pregnancy itself may have been exaggerated, yet a large family may have indirectly a bad effect upon the mother in that it must almost certainly mean less opportunity for rest and for that careful regulation of her life which is so essential for the maintenance of arrest, and among the poor too often also insufficient food.[4]

Hæmoptysis.—In speaking of the treatment of hæmoptysis, we have in mind the causes of hæmorrhage already indicated as operative in phthisis—viz., active hyperæmia, diseased small vessels, and erosion or aneurismal dilatation of large vessels. The two former conditions account for most attacks of primary hæmoptysis and for such intercurrent attacks as are preceded by, or attended with the symptoms and signs of fresh accession of lung disease; the latter condition being answerable for the severe and sometimes fatal hæmorrhage proceeding from cavities, most commonly in the later stages of the disease.

Primary and Intercurrent Hæmoptysis.—Shock is a marked symptom in early hæmoptysis, especially in first attacks. The agitation of the patient must be calmed by the confident assurance of the absence of immediate danger, and all measures of treatment should be carried out without bustling about or whispering. The doctor will often treat his patient best by quieting anxious friends. The recumbent posture, with head and shoulders slightly raised by pillows, should be adopted, with light coverings and warmth to the feet. Ice should be given, and all stimulating restoratives strictly forbidden. Indeed, the early faintness which so commonly supervenes, when short of actual syncope, should be encouraged rather than prevented, since it involves a low tension in the pulmonary system, a condition favourable to the formation of a clot, which may close the bleeding vessel.

In all cases of hæmoptysis the aspect of the patient and character of pulse should be noted, the temperature taken, and the stethoscope lightly applied over the front of the

chest, no extra effort of breathing being allowed; the heart's sounds should also be carefully listened to. All these observations can be made without disturbing the patient in the least, and information as to the attack can be gained from friends.

There are different types of cases to be observed, for which different plans of treatment are indicated. In a considerable proportion of cases there is pallor, with drawn features, chilly surface, and small, thready, quick pulse, significant of combined nervous shock, loss of blood, and constitutional feebleness. In such cases opium is of great value, a small hypodermic injection of morphia rapidly calming the agitation and quieting the heart's action. It must be administered, however, with caution, and not in sufficiently large doses to check all cough, lest the danger of broncho-pneumonia from the retention of infected material be aggravated. The bowels should be attended to, and later may, if necessary, be kept freely open, the pressure of the blood in the general and also in the pulmonary circulation being thus controlled. For this purpose a drachm of sulphate of magnesium, three or four times a day, may be prescribed. For some days the patient must be kept absolutely at rest in bed, not being allowed to rise on any pretext, and not encouraged to talk by the visits of sympathising friends. His food should be light, and should consist of two to three pints of cold, or at least only just warm, milk each day, with a little bread and butter, junket or custard. If he desires it, he may be allowed some ice to suck.

Under this treatment the hæmorrhage will often cease; should it continue, other remedies must be considered, but in gauging their value we must remember that cases of primary hæmoptysis as a rule subside without much trouble, and any remedy used in a sufficiently large number of cases will gain a reputation. Salt and water was long held by the Brompton nurses as of great efficacy.

Of the special remedies of value, the *nitrites* may first be mentioned. The inhalation of amyl nitrite leads to the dilatation of the peripheral vessels, and thus to a lowering of tension in both general and pulmonary systems. So much is this the case that in the experiments upon dogs of MM. Pic and Petitjean[5] the lungs became white and anæmic within two or three minutes of the amyl nitrite injection, and according

to these observers, if a cut was made in the organ at the moment of the injection, the copious hæmorrhage resulting was stanched as though by the application of a ligature. The effect was said to last about five minutes. Amyl nitrite is thus usefully inhaled at the first onset of the hæmorrhage, the anæmia of the lung and the fall in pulmonary pressure assisting in the formation of a clot, and sealing the bleeding point. A patient who has had hæmoptysis may be advised to carry the capsules in his pocket, to be used in the event of any emergency arising. The effect is, however, transitory, and if the blood-pressure is estimated to range above the normal, two-grain doses of sodium nitrite may often be usefully employed to ease it to a lower level. Erythrol tetranitrate or nitro-glycerine may be prescribed for the same end.

In certain cases five-grain doses of *ipecacuanha*, repeated every one or two hours for a few doses, as recommended by Sir Robert Philip, prove valuable. We have ourselves never been tempted to adopt Trousseau's plan of giving an emetic to arrest hæmoptysis.

Oil of turpentine is another remedy which is often found useful, although its mode of action is not clear. It should be given in twenty-minim doses every four hours, and may be combined with three minims of oil of cloves, two drachms of mucilage of acacia, and cinnamon water to one ounce, care being taken to examine the urine from time to time during its administration for the presence of albumin.

Many other drugs have been recommended for hæmoptysis. Thus, *calcium chloride* and *calcium lactate*, in doses of ten to fifteen grains three times a day, have been thought by some observers to increase the coagulability of the blood, and to be thus indicated. We have tried them on many occasions, and in more frequent and larger doses than the above, but have not convinced ourselves of their utility. In former days tannic and gallic acid and acetate of lead were also much employed, but a study of their pharmacological action renders it more than doubtful whether they possess that remote astringent and hæmostatic effect with which they were once credited. Their astringent action on the bowel is directly opposed also to the requirements of the case. We have found tincture of hamamelis (♏xl.) in

combination with a like dose of hydrobromic acid every six hours of a certain value in cases of hæmoptysis when the first outburst has passed.

Another remedy which formerly enjoyed a great reputation is *ergot*, given at first in a full dose (one or two drachms of the liquid extract in iced water), and twenty to thirty minims every hour afterwards for a few doses. The action of this drug is to stimulate organic muscle, including that of the arterioles; but whilst thus constricting small vessels, it at the same time, as shown by the late Dr. Andrew, raises both pulmonary and systemic blood-pressures, and any good effect upon the pulmonary arterioles is thus to some extent counteracted. Nevertheless, we have known it apparently instrumental in arresting hæmoptysis in cases in which other remedies had failed.

More recently *adrenalin* has been advocated with the idea of constricting the pulmonary vessels, and thus closing the bleeding point; but Drs. Brodie and Dixon[6] have shown it to be not capable of producing this result, and Dr. Langdon Brown[7] records that "in an animal killed by a fatal dose, while all the other tissues were anæmic—from the general peripheral constriction—the coronary vessels were distended with blood, and the lungs showed a condition of the most intense congestion, being a deep plum colour." Its use is therefore contra-indicated.

The continuous application of ice to the chest for early or intermittent hæmoptysis, a remedy much used, is in our opinion to be deprecated. We have never observed it to do good, and have seen cases in which it appeared to do much harm.

For the hæmoptysis which sometimes occurs in persons with venous plethora and males of intemperate habits, and which not uncommonly ushers in a rapid inflammatory form of phthisis, a drachm of sulphate of magnesium, with fifteen minims of aromatic sulphuric acid, may be prescribed with advantage every four hours until the secretions become free and watery, and in some cases it is well to begin with a mercurial. In very exceptional cases of this kind depletion from the arm may be advised.

In patients in whom there is distinct evidence of syphilitic cachexia full doses of iodide of potassium, with or without

mercury, should be combined with the more usual methods of treatment. In these cases the hæmoptysis is sometimes very severe, and yet yields readily to treatment.

In the early stages of phthisis, as we have seen, active inflammatory hyperæmia is commonly present, and may give rise to hæmorrhage, which does not usually amount to more than a coloration or streaking of the sputum. The recumbent position of the patient during sleep is a factor tending to increase the hyperæmia, and if the head and shoulders be raised to an angle of 45 degrees with the trunk, the hæmoptysis will in some cases cease.[8]

Slight staining of the expectoration, or even a small quantity of fresh blood, not infrequently appears during the eliminative periods of the pneumonic forms of phthisis. Such occurrences need occasion no modification of treatment, being due to the breaking across of trabecular vessels of small size, already partially occluded. As a matter of precaution stricter quietude must be enjoined for a short time after any such occurrence. Mixed sanguineous expectoration in cavity cases is best combated by free counter-irritation by the application of a blister, and subsequently dressing the surface with savin ointment for a week or ten days.

Summary.—In an ordinary case of primary hæmoptysis our treatment, therefore, would be to reassure the patient and his friends, to give a dose of morphia, and later some such slight astringent as tincture of hamamelis, in combination with hydrobromic acid in iced water. If amyl nitrite be at hand it should be inhaled during the attack. Should the hæmorrhage be more severe and repeated, small doses of nitrite of sodium may be given for a couple of days to ease the blood-pressure. In other cases in which the hæmorrhage continues, whilst the blood-pressure is low, turpentine or ergot may be prescribed. The patient should be kept at absolute rest, and the diet should consist of cooled liquids, and such semi-solids as custard, blancmange, and bread and butter. We should soon, however, begin cautiously to feed our patient, avoiding only hot fluids. It is of the utmost importance that the bowels should be kept clear, and in certain cases attended with plethora sulphate of magnesia and dilute sulphuric acid are the best medicines. Under this treatment the hæmorrhage is as a rule quickly controlled, and we have never known it

necessary in the class of case which we are now considering, to resort to artificial pneumothorax.

Recurrent Hæmoptysis.—In the treatment of this form of hæmoptysis, which, it will be remembered, is generally due to the rupture of an aneurism in a pulmonary cavity, the same general principles which we have already sketched out must be adhered to. Absolute rest must be insisted upon, cough and excitement allayed by opium, and arterial tension reduced by nitrites, salines, or the other remedies to which we have alluded. In this way Nature is assisted in forming a coagulum at the seat of rupture, and the hæmorrhage is checked.

The tendency to recurrence of hæmorrhage in these cases must be remembered, and care must be taken not to allow the patients to move about too soon. In those patients, too, who are gifted with rapid blood-making powers, and who pick up flesh with great rapidity after hæmoptysis, a judicious abstinence from butcher's meat and the complete withdrawal of stimulants may ward off or postpone the next attack.

The hæmorrhage in this form is far more profuse than in primary hæmoptysis, and is often immediately fatal. In some instances, as in that of the patient whose case we have recorded (p. 553), it recurs again and again, until the patient arrives at the lowest ebb compatible with life, when the bleeding vessel is stanched by coagulum, and recovery commences. In some other cases, however, the patient dies suddenly from combined syncope and blocking of the air-passages, with the expectoration of only a very small amount of blood.

It is in this class of case, in which the hæmoptysis is severe and repeated, and the patient's life in danger, that treatment by artificial pneumothorax is of value (see Chapter LI.). By this means the lung is collapsed, pressure is exerted upon the bleeding point, and the formation of clot facilitated. We have known several cases thus successfully treated, and have no hesitation in advising the performance of the operation provided we can be reasonably satisfied, from the physical signs and symptoms, as to the side from which the hæmorrhage is proceeding. It sometimes happens, however, that with advanced disease in one lung, the patient's sensations tell him that the blood is coming from the other and less affected lung, and this is not impossible, for in rapidly advancing cases we have known aneurism to form in the lung much less exten-

sively diseased. Under such circumstances the treatment must be abandoned, the extensive involvement of the other lung forbidding us to entertain the idea of compressing the more healthy side.

With reference to prophylactic treatment, patients suffering from phthisis, who have chronic cavities, and have had warnings of hæmoptysis, should be cautioned against unnecessary and especially sudden muscular efforts calculated rapidly to raise the blood-pressure, and should therefore avoid hurrying upstairs, walking fast, hastening to catch trains, and so forth.

Spurious Hæmoptysis. We have dealt with the treatment of this non-pulmonary form of hæmorrhage in the chapter devoted to its description (see p. 557).

Vomiting with Cough.—This distressing symptom is especially characteristic of the more indurative forms and stages of the disease, and its successful treatment is attended with much difficulty. The symptom is primarily due, as we have pointed out (p. 516), to the mechanical difficulty in expelling secretions from cavities and bronchial tubes which are surrounded by dense, tough, airless consolidations; and, secondly, it is distinctly to be observed in many cases that there is undue irritability of the vagus, giving rise to cough directly food is taken into the stomach. Any catarrhal condition of this organ must be treated by alkalies, bismuth, and hydrocyanic acid taken an hour before meals, and even in the absence of catarrh this alone may relieve the symptom by rendering the mucous membrane less responsive to nervous influences. A mixture containing five grains of Alum, five minims of Liquor Potassæ, and Peppermint water to an ounce, is also sometimes helpful in these cases. If there appears to be a hyperæsthetic condition of the vagus, a little hydrocyanic acid with ten-minim doses of the Liquor morphinæ hydrochloridi, taken half an hour before the principal meals, either alone or with bismuth and soda, may succeed in preventing the cough. In other cases, again, we have found a course of strychnia as a nerve tonic of value.[9] The patient should keep strictly at rest for an hour or so after meals.

Intercurrent Pleurisy.—Intercurrent pleurisy at the upper half of the chest is best treated by the application of small blisters or the Liquor Epispasticus. If the pain be rather of

a dull aching than of a sharp pleuritic character, and if physical signs point to the presence of fresh pulmonary trouble, the more gradual effect of iodine applications is to be preferred. The Tinctura iodi fortis should be painted over the part each day or second day for two or more applications, a layer of pine-wool covered with oiled silk being kept constantly applied over the part painted. It must be remembered that some skins are much more sensitive to iodine than others, and in such cases, and especially in children, the Tinctura iodi mitis will be found sufficiently strong.

When some portion of the lower half of the chest is affected with pleurisy in the course of phthisis, the prompt application of a long piece of Leslie's strapping three or four inches broad, so as to extend round the affected side to a couple of inches beyond the median line in front and behind, will, by restraining respiratory movements, at once relieve pain, and often arrest the local inflammation. This treatment is of great value in many cases, and is to be particularly advised when, in addition to the signs of pleurisy, superficial crepitations with a few liquid clicks suggest that liquefaction of caseous pneumonic centres is proceeding close under the pleura. The timely application of strapping in such cases may, by relieving the lung from the shock of cough, avert pneumothorax. Some patients cannot bear this application, however, and the rough clinical test of its probable usefulness in any given case is to hold the side with steady pressure of the hand, and see whether relief from pain is thereby obtained. When strapping is not tolerated, the application of a few leeches, or a small blister, with a hot linseed poultice over it, will speedily give relief, and this line of treatment may be followed with advantage when the pain is severe.

Tuberculous Meningitis.—The early stages of tuberculous meningitis are, as already pointed out (p. 563), very insidious and obscure, and it must be confessed that the progress of this fatal malady is beyond our control. Some of its more distressing symptoms may, however, be mitigated. A brisk calomel purge should first be given, and if pain in the head be a marked symptom, it will be relieved by the application of cold lotions or the ice-cap. In severe cases relief will be afforded by the application of leeches to the temples. The

room should, in the earlier stages of the disease, be darkened; care must be taken to relieve the bladder if necessary.

Full doses of bromide and smaller doses of iodide of potassium seem sometimes to give relief. When twitchings are present, chloral should be given in combination with the bromide. Should convulsions threaten, the chloral should be sufficiently pushed to avert, if possible, this symptom, so distressing to friends, although, happily, unfelt by the patient, the remedy being administered in a little water or barley-water by the rectum, if it cannot be taken by the mouth. A few whiffs of chloroform sometimes appear to restrain convulsions. Considerable relief may be given in many cases by lumbar puncture, the excess of fluid effused into the ventricles being thus drained away, and pressure symptoms for a time relieved. The tapping should be repeated if necessary.

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CHAPTER L

SPECIFIC TREATMENT OF PULMONARY TUBERCULOSIS

We have in the preceding chapters considered the treatment which should be adopted in dealing with the various forms of pulmonary tuberculosis. Apart from the combating of special symptoms, the lines of treatment suggested are, broadly speaking, designed to shield the patient as far as possible from the absorption of excessive doses of toxines, whilst at the same time his vitality is stimulated and his strength sustained, and the formation of protective substances in the blood and tissues thus increased. We have now to discuss whether there is any specific treatment which will play a more direct part in our fight against the disease.

The inhalation of bactericidal sprays, heated air, stable emanations, compressed and rarefied airs, sulphuretted hydrogen, the administration of sulphuretted waters and gases by the mouth and *per rectum*, the subcutaneous injection of antiseptic drugs of all sorts, have been tried by able and sanguine observers, but the results obtained have not been such as to warrant our dwelling any further upon them. Many of these remedies may take a humbler place in the treatment of particular symptoms, but none have the slightest value as specific measures against the malady.

The fundamental object in the treatment of tuberculosis is, as already explained, to obtain immunity to tuberculous activity by fortifying and maintaining resistance to further attack. Vaccines are employed in acute and chronic diseases of infective origin with the object of securing immunity by increasing opsonic and other antimicrobial means of resistance and stimulating phagocytic activity. It is doubtful, however, whether they are of value in the acute phase of any illness, such as pneumonia, scarlet fever, typhoid, tuberculosis, etc.,

for in this phase of such diseases the blood is already overwhelmed with the toxines from the microbic activity responsible for the disease. Before exposure to infection, however, and possibly during the incubation period of some infective diseases, an appropriate vaccine may be of value by artificially stimulating resistance to the toxine in question. In some chronic infections also, when resistance has reached a low ebb, a stimulating reaction may be thus provoked with advantage.

Tuberculosis is a malady in which, during its acute phases, the system is already more than adequately charged with toxines from the bacillus. In the sub-acute and chronic stages, again, there are always patches of sub-active bacillary lesions or centres of quiescent disease, for the most part not walled off from the general lymphatic system, so that the patient, if we may so express it, is rather "over salted" than otherwise, and it is only necessary to flush the centres of disease by an excessive activity of circulation through exercise, mental excitement or anything that hurries the breathing and heart's action, to produce such an extra absorption of tuberculous toxine from the lesions as shall cause a reactive rise of temperature.

Thus is explained the importance in tuberculosis of rest treatment when the disease is active, and graduated exercise treatment when the disease is passive; and such are the grounds on which doubt is cast on the general utility of tuberculin in this disease, and on which an insight is obtained as to how immunity is, in many cases, acquired.

There are, however, exceptional cases of pulmonary tuberculosis and of local tubercle situated in other parts than the lungs—as, for example, in bones, in glands and in other positions—in which tuberculin may be employed, and the following are, briefly, in such cases the methods to be pursued:

Varieties of Tuberculin.—Many varieties of tuberculin are now to be obtained which differ from one another in their mode of preparation and in their chemical features. Clinically, however, the results obtained from their employment are not very dissimilar. The more important are the following:

1. *Old Tuberculin—Tuberkulin Alt.*—This was the preparation used by Koch in 1890 in the treatment of the patients

who thronged to Berlin in that memorable year. It is prepared from a culture of human tubercle bacilli which have been grown for some six weeks in 5 per cent. glycerine broth. This is boiled for one hour to kill the bacilli, and the fluid concentrated to one-tenth of its volume by evaporation at a temperature not exceeding 70° C. The bacilli are then separated off by filtration. The golden-brown, somewhat viscid liquid thus obtained is Old Tuberculin, which contains the toxines which are soluble in 50 per cent. glycerine. One c.c. of this preparation is regarded as equivalent to one gramme (1,000 mgr.).

2. *Albumose-Free Old Tuberculin* (*A.F.*).—This resembles Old Tuberculin in all respects except that it is grown upon a proteid-free medium, contains no albumose and is believed therefore to be less toxic in its effects.

3. *Denys' Tuberculin—Bouillon Filtré* (*B.F.*).—This differs from Koch's Old Tuberculin only in the non-concentration of the broth culture. The culture of human tubercle bacilli at the end of six weeks' growth is filtered, and the unboiled, unheated filtrate constitutes Denys' tuberculin. Professor Denys believes that in this way deterioration of the toxines is prevented.

4. *New Tuberculin—Tuberculin Rest* (*T.R.*).[1]—This is prepared by grinding dried human tubercle bacilli in an agate mortar, extracting with distilled water, and centrifugalising. The upper layer, called by Koch "T.O." (*Tuberculin Oberst*), closely resembles Old Tuberculin. The lower layer, or remainder (*Rest*), which thus contains the pulverised and partially extracted bodies of the bacilli, is suspended in water containing 20 per cent. glycerine to prevent decomposition, and constitutes New Tuberculin, or T.R. Each cubic centimetre contains 2 milligrammes of solid matter derived from 10 milligrammes of dried bacilli. T.R. thus differs materially from Old Tuberculin, which contains, as we have seen, not the bacillary bodies, but those substances derived from them, which are soluble in 50 per cent. glycerine.

5. *New Tuberculin—Bacillary Emulsion* (*B.E.*)—This was introduced by Koch in 1901, and consists of pulverised human bacilli, not extracted in any way, but suspended in equal parts of water and glycerine. Each cubic centimetre of the preparation contains 5 milligrammes of solid matter.

In addition to the above, we may mention the *Sensitised Bacillary Emulsion* (*S.B.E.*) and *Professor Béraneck's Tuberculin*, which, with many other varieties of tuberculin, prepared in different ways, some from human and some from bovine strains of bacilli, contain different proportions of the intra- and extracellular toxines.

Although differing in their composition and in their strength, all varieties of tuberculin have this in common, that they contain undoubted toxines, and that by their use an attempt is made to call forth an active response on the part of the organism and an increased production of protective substances. The process aimed at, in fact, is the production of an *active immunisation*, in which the cells of the body must play their part, as opposed to the *passive* rôle assigned to them when a poison within the body is neutralised by the introduction of an antitoxic serum.

Of the above varieties of tuberculin the New Tuberculin (T.R.) and Denys' Tuberculin are the milder preparations. With all, however, the clinical effects are not very dissimilar, and in each case our aim should be, by commencing with very small doses, and by increasing the dose very gradually, to avoid the production of any clinical "reaction," whether indicated by fever, malaise, pains in the joints or back, increase of cough and sputum or other manifestation. We may add that New Tuberculin (T.R.) is a much more expensive preparation than the Bacillary Emulsion, and on this ground the latter is to be preferred.

Dosage.—Tuberculin is best given by subcutaneous injection, ·01 c.mm. ($\frac{1}{100000}$ c.c.) of Old Tuberculin, A.F., T.R., or B.E., being a common dose with which to commence. The following scheme of dosage, on the lines suggested by Drs. Bandelier and Roepke,[2] we have found useful as a guide in practice, it being understood that in the event of any clinical reaction occurring a longer interval than usual must be allowed before the next dose, which should also be somewhat smaller than the dose which caused the reaction. Should no reaction follow the reduced dose, the amount of tuberculin should again be gradually increased. If the doses are increased too rapidly, a condition of hypersusceptibility ("anaphylaxis") to the tuberculin may be developed, which it is important to avoid.

SCHEME OF DOSAGE FOR THE ADMINISTRATION OF TUBERCULIN (O.T., A.F., T.R., AND B.E.) BY SUBCUTANEOUS INJECTION.

An injection to be given every alternate day, commencing with ·001 c.mm. (a millionth of a c.c.) and proceeding as follows:

·001, ·003, ·006 c.mm.
·01, ·03, ·06 c.mm.
·1, ·15, ·2, ·3, ·5, ·7 c.mm.

Then continue with an injection twice a week.

1, 1·5, 2, 3, 5, 7 c.mm.
10, 15, 20, 30, 50, 70 c.mm.

Finally, if no contra-indication exists and time allows, an injection to be given once a week as follows:

100, 150, 200, 300, 400, 600, 800, 1,000 c.mm. (1 c.c.).

The maximum dose may be repeated several times at increasing intervals.

Cases suitable for Treatment.—There can be little doubt, both on the theoretical grounds already hinted at (p. 712), and in view of the practical results obtained at Midhurst, to which we shall shortly refer, that the value of tuberculin as a remedial agent in the treatment of phthisis has been greatly overestimated. It may, however, still be prescribed in exceptional circumstances. It may, for example, be given, when other treatment has failed, in quiescent apyrexial cases in which improvement has proceeded up to a point and then come to a halt, or in cases in which, in spite of a stationary and fair weight, and an absence of pyrexia, the pulmonary disease slowly and insidiously extends. It must be remembered that in order to produce a good result tuberculin must call forth an active immunising response on the part of the patient, and a certain degree of vitality is therefore essential for its successful administration. *In febrile cases the remedy is contra-indicated*, the reactive power of the patient being already stimulated to the full by the excessive doses of toxine which are being absorbed.

Results of Treatment.—In attempting to decide upon the value of tuberculin in the treatment of phthisis, we must at once recognise that great caution must be exercised in basing an opinion upon individual cases. It has been our lot from time to time when treating cases in the wards to be struck with the improvement shown by a patient receiving tuberculin, and we have been inclined to attribute the result

to the specific treatment, until a few beds farther on we have met with a patient in an almost similar condition untreated by tuberculin, in whom the improvement has been equally rapid. Had the first patient been the only one under observation, it would have been difficult to avoid overestimating the possible value of the remedy. Our experience is not exceptional. The capacity for improvement under better conditions of feeding and hygiene is a marked feature in many phthisical cases, and we must accordingly gauge the result of treatment not so much by impressions derived from individual patients, as by a careful inquiry into the effect produced in a large series of cases, and by the permanency of the results obtained.

In this connection we must refer to the careful investigation in regard to the value of tuberculin treatment recently made on actuarial lines at the King Edward VII. Sanatorium, Midhurst, by Dr. Bardswell and Mr. Thompson.[3] These observers compared the results obtained in 384 male and female patients belonging to groups 1 and 2 (incipient and moderately advanced cases), who had tubercle bacilli in the sputum, and who were treated at the sanatorium over a period of three years, during which sanatorium methods only were employed, with a series of 352 comparable cases admitted during the three succeeding years, when tuberculin was also being administered. Of these 352 cases 238 received a course of tuberculin in addition to sanatorium treatment. If tuberculin were an efficient aid to treatment, its value should be shown by an improvement in the general results obtained. The tuberculin used, we may add, was almost exclusively Albumose-Free Old Tuberculin (A.F.) and Koch's Bacillary Emulsion (B.E.), and the method of administration that of Bandelier and Roepke, and in the last year the reactionless method of Sahli and Trudeau, both of them methods of recognised repute.

The results obtained were as follows:

(1) *Immediate Results.*—The condition on discharge of the patients belonging to the two series was practically the same, the percentage of those recorded as "arrested or much improved," "improved," "stationary or worse" being very similar.

(2) *Sputum Records.*—The percentage of patients in whom

tubercle bacilli disappeared from the sputum during treatment in the sanatorium showed a close agreement in the two series, 26·3 per cent. of the patients during the control period, who received sanatorium treatment only, losing their tubercle bacilli, as compared with 26·7 per cent. of those treated during the tuberculin years. This point is of importance since, as we have indicated elsewhere (p. 631), patients who lose their bacilli during treatment at the sanatorium have a better prognosis than those in whom tubercle bacilli persist.

(3) *Subsequent Results.*—With regard to the *after-histories* of the patients, compared in each case up to four years after discharge, it was found that the ratio of "actual" to "expected deaths" was 18·0 per cent. for the patients who during the control period received sanatorium treatment only, and 18·1 per cent. for those treated during the tuberculin period.

These results show that tuberculin, when given in the manner adopted at Midhurst, and as an adjunct to sanatorium treatment, had no appreciable effect, either favourable or unfavourable.

It is interesting to note that Messrs. Elderton and Perry,[4] after an actuarial study of the valuable data from the Adirondack Sanitarium, in New York State, arrived at a somewhat similar conclusion—namely, that, while it was not possible to say that there was no case in which tuberculin might not be of use, its value as judged from the subsequent mortality of the patients had not been proved.

It is clear, therefore, that tuberculin can no longer be regarded as having the specific value at one time attributed to it, and its employment, as we have already indicated, must be correspondingly restricted.

Serum-Therapy.—In addition to the method of active immunisation aimed at by the treatment with tuberculin, an attempt has been made to effect a passive immunisation by means of serum-therapy, whereby protective bodies prepared in other animals are brought to the immediate assistance of the patient. Of such sera the best known are those of Maragliano and of Marmorek. Neither have, however, fulfilled the hopes with which they were introduced, and they are now but little used.

The Specific Treatment of Mixed and Secondary Infections.—In an earlier chapter (p. 467) we have given reasons for be-

lieving that in certain rapidly progressing cases of phthisis the fever and the spread of the disease are due in part to the agency of organisms other than the tubercle bacillus, and attempts have been made, by isolating such organisms from the sputum and producing appropriate vaccines, to cut short their activity, to the relief of the patient and the improvement of his symptoms. The results have, however, been disappointing, and, as we have already pointed out, but little must be hoped from vaccines in the acute stages of the disease, when the body is already receiving excessive doses of the microbial poisons. More perhaps may be expected in chronic cases in checking excessive cough and bronchial excretion, which, as we have seen, are often the result of secondary infection by pathogenic organisms.

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CHAPTER LI

TREATMENT OF PULMONARY TUBERCULOSIS BY (1) ARTIFICIAL PNEUMOTHORAX, (2) SURGICAL INTERVENTION

Artificial Pneumothorax.

In the preceding chapters we have discussed the hygienic and climatic treatment of pulmonary tuberculosis, the use of tuberculin, and the value of drugs. In certain cases of a fairly well-defined kind, in which, in spite of all efforts, the disease makes progress, and especially in some in which recurrent hæmoptysis is a well-marked feature, the question of inducing an "artificial pneumothorax" presents itself, and must now be considered.

By this method air or gas is introduced under strict antiseptic precautions into the pleural cavity by means of a special apparatus, and the lung is collapsed, thus rendering it passive, allowing active lesions to cicatrise and heal, while at the same time absorption of toxine is lessened. If there has been hæmorrhage, the pressure tends to prevent its recurrence. In the earlier days of the treatment nitrogen was the gas employed, but experience has shown that it possesses no advantage over air, which is not absorbed more quickly, and air is now commonly used. The practice of introducing oxygen at the first injection, with a view to diminish the risk of gas embolism, appears to be based upon a misconception and to possess no practical advantage.

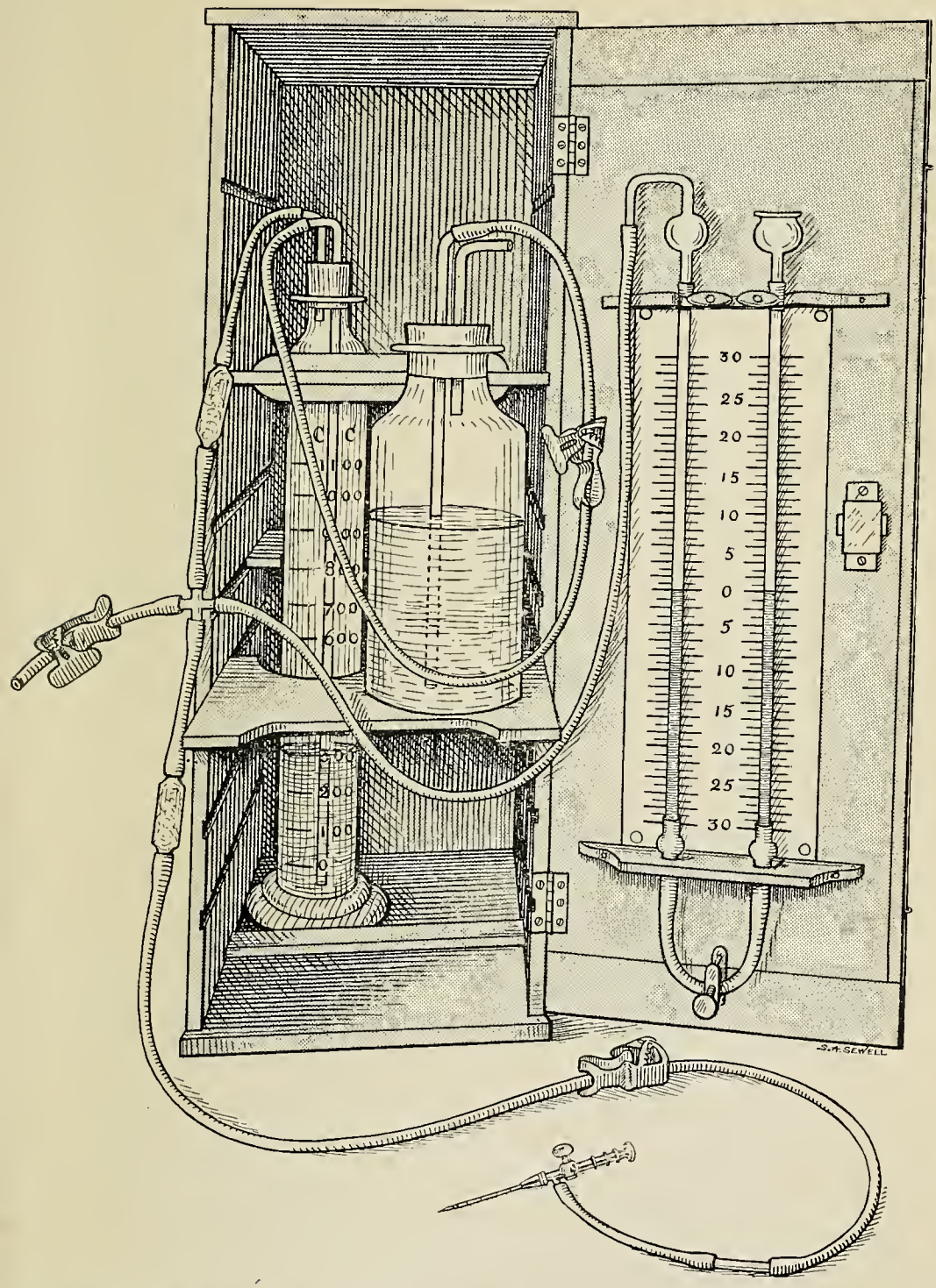
The experiment was apparently first made by the late Dr. Cayley in the year 1885 in a case of hæmoptysis, to which we have already referred, the pneumothorax in this instance being produced by incision through the chest wall. For the modern developments of the method and its present technique we are indebted especially to Professor Forlanini of Pavia, Professor Brauer of Hamburg, and Professor Saugman of

Vejlefjord in Denmark, and in this country to Dr. Clive Riviere,[1] to whose work we may refer the reader for many technical details.

Technique.—For the introduction of the air some simple portable apparatus is required. Several have been introduced, among which we may mention that of Dr. W. Parry Morgan,[2] but the one devised by Dr. Lillingston and Dr. Vere Pearson[3] may be recommended as simple and effective, and, with some slight modifications, is now in use at the Brompton Hospital (Plate XXXIII.). It consists of a needle connected by rubber tubing to a bottle containing air, the "gas bottle," and by means of a cross-piece with a manometer, whereby the pleural pressure is measured. The gas bottle itself is connected with a "pressure bottle" containing a coloured antiseptic solution, such as 1 in 1,000 perchloride, by the lowering or raising of which the outflow of gas into the pleural cavity can be regulated. Between the needle and the cross-piece connecting with the manometer a piece of glass tubing containing sterilised cotton-wool for filtering the air is inserted, and a second between the cross-piece and the gas bottle. It is well also to insert, as we have done in the figure, a short length of glass tubing not far from the needle, so that when the instrument is used for gas-replacement (p. 114) the entry of fluid into the tubing may at once be detected, should this occur. The fourth limb of the cross-piece, to which in Plate XXXIII. a short piece of rubber tubing with clamp is attached, has been added at the suggestion of Dr. L. S. T. Burrell to facilitate refilling of the gas bottle with air, should this be necessitated during the progress of the operation.

For the initial operation and for the first few reinflations the patient must be in bed. A suitable site for the puncture is to be found in the lower axillary region, as, for example, the sixth space in the mid-axillary line, since here, as we have pointed out in dealing with paracentesis thoracis, the parietes are thin and the intercostal spaces roomy, and adhesions are less likely to be present. If, however, adhesions prevent the introduction of air, attempts must be made in other regions in the lower part of the chest, such as the eighth or ninth space below the angle of the scapula, where physical signs indicate but little disease. If three or

PLATE XXXIII



APPARATUS FOR PERFORMING ARTIFICIAL PNEUMOTHORAX DEVISED BY DR. VERE PEARSON AND DR. LILLINGSTON (SLIGHTLY MODIFIED).

NOTE.—To facilitate the reading of the intra-pleural pressure, the manometer scale is graduated in half centimetres: the rise or fall of the water in the limb exposed to the air being accompanied by a similar fall or rise in the other limb, the figures give the pressure in c.c. of water.

To face p. 720.

four punctures prove fruitless, owing to the presence of widespreading adhesions, the operation must be abandoned. Professor Saugman[4] met with failure in 43 of his 138 cases.

The selection of the pneumothorax needle is of importance. At first a pointed needle was used, but with this there is some danger of wounding the lung, with the possibility of gas embolism when the air is introduced, and there can be no doubt that an instrument such as that devised by Dr. Riviere[1a] is better for the initial operation. This consists of a trocar and cannula, the latter of 1·8 millimetres (·072 inch) gauge, with a side-opening near the tip, and with a sharpened cutting end. The trocar and cannula are carefully introduced to a depth of about ¾ centimetre, when the shoulder of the cannula will have passed through the skin. The trocar is then withdrawn and the tap of the needle closed. The cannula with its sharpened circular end is slowly pressed onward until the pleura is reached and the sudden "snap" is experienced, which indicates that the parietal pleura has been perforated. This will be confirmed by the sudden appearance in the manometer of a negative pressure of some 10 centimetres of water, and a definite respiratory fluctuation of from 4 to 8 centimetres. The depth at which the pleura is situated will naturally vary with the thickness of the parietes; in thin subjects it may be only 1 centimetre from the surface, and Professor Saugman has never found it at a greater depth than 3 centimetres.

When it is thus made clear that the needle is in the pleural cavity air may be introduced. On the first occasion about 400 c.c. are often sufficient, but the amount must depend to some extent upon the size of the chest and the ease with which the lung collapses. At the end of the first inflation the intrapleural pressure should still be slightly negative. When, however, the operation is done to check hæmoptysis, 1,000 c.c. should at once be introduced. The air at first is somewhat quickly absorbed from the pleura, and further inflations must be made at intervals of a few days, later at intervals of a week, then a fortnight, and at last, when the pneumothorax is well established, at intervals of four to eight weeks or even longer. The amount of air introduced at each refill is gradually increased, so that at the fourth or fifth inflation, when the lung is completely collapsed, the intrapleural pressure at the

termination of the operation should measure about + 5 centimetres of water. The object of the treatment is to obtain, without undue stretching of the mediastinum and without discomfort to the patient, as complete a collapse of the lung as possible, so that when examined by the X-rays it may be seen lying by the side of the vertical column. The dates of further refills, and the quantity of gas introduced, should be controlled when possible by noting on the screen whether the lung remains collapsed or shows a tendency to re-expand. The presence of bands of adhesions, which prevent complete collapse of the lung and militate against the efficiency of the treatment, are also brought into evidence by X-ray examination. For the reinflations the trocar and cannula may be again used, or, if desired, a pointed needle, such as that devised by Professor Saugman, since the needle will now enter a cavity already containing air, and there is not, therefore, the same risk of wounding the lung as at the first operation. The needles should be kept in absolute alcohol and dried before use by passing through the flame, thus rendering them aseptic and insuring that the lumen is free from fluid, and capable at once of registering the intrapleural pressure.

The performance of an artificial pneumothorax is not entirely free from risk, since occasionally, at the moment of perforating the pleura, the patient becomes faint and the pulse and respiration irregular, and in a few cases such an attack has terminated fatally, death being attributed to reflex spasm of the cardiac or cerebral vessels. To this condition the terms "*pleural reflex*" and "*pleural shock*" have been applied, and its occurrence has been noted in other manipulations of the pleura, notably irrigation, and for this reason we have advised against this procedure save in very exceptional circumstances (p. 129). Experimental evidence suggests that the danger of pleural reflex is diminished by proper anæsthesia, and in performing artificial pneumothorax it is well always to give a *sedative*, such as an injection of morphia (gr. $\frac{1}{4}$) or omnopon (gr. $\frac{1}{3}$) *half an hour before the initial operation, and carefully to anæsthetise the site of puncture right down to the pleura* with a sterilised 2 per cent. solution of novocaine in normal saline. At the refill the sedative is not usually needed, but the local anæsthetic should be employed.

The danger of *gas embolism* from the introduction of air into a vessel of the lung has with improving technique been largely eliminated, and the accident should be of the greatest rarity,[5] provided that care be taken never to introduce the air until the negative pressure and the respiratory excursion, as registered by the manometer, prove that the needle is free in the pleural cavity.

Duration of Treatment.—How long the treatment should continue and the compression of the lung be maintained is a matter in regard to which there is some difference of opinion, and there is at the present time a tendency to lengthen the duration of treatment. It is generally felt, however, that eighteen months to two years is a wise minimum, and that in more serious and advanced cases this may be extended to three or even four years, inflations being given at the later stages at intervals of six to eight weeks or even longer. During the period of compression the lung undergoes some degree of fibrosis, and it is possible that it may not fully expand when the treatment is discontinued, so that the chest falls in to some extent.

Complications.—During the course of treatment two complications may be met with which we must now consider. These are pleurisy and perforation of the lung.

1. *Pleurisy.*—This is a common complication of artificial pneumothorax, and occurs, it is estimated, in nearly half the cases, especially those in which the disease is acute in character or advanced in degree. It is usually attended with effusion, which is as a rule serous in character, sterile on culture, and containing tubercle bacilli. Later the fluid sometimes becomes purulent.

The complication occurs most commonly within the first three months; in other cases at some later period during the course of treatment, possibly not until the third or fourth year. The symptoms are sometimes very slight, but in other cases the onset is sudden, with pyrexia and pain in the side, the temperature remaining raised for two or three weeks or more, and then falling gradually to normal, as in the case of a simple pleurisy with effusion. The rapid effusion of fluid in such cases may raise the intrapleural pressure and embarrass the respiration, and thus necessitate the withdrawal of some of the air; but in our experience the occurrence of

effusion, when the febrile stage has passed, is often helpful rather than the reverse to the patient, by tending to keep the lung collapsed and thus allowing of refills at longer intervals.

2. *Perforation or Rupture of the Lung* is fortunately an event of rare occurrence, though we have seen at least one instance, and several are recorded by Dr. Sachs,[6] Drs. Marshall and Craighead[7], and other observers.[5] It is apt to occur where adhesions prevent more than a partial pneumothorax, and the lung is thus stretched and unprotected. It is perhaps surprising that the accident should not occur more often in such cases, when it is remembered that the pleural surfaces are deliberately separated by the air introduced, and thus the formation of adhesions over patches of advancing disease, which so often prevent pneumothorax, is no longer possible. The accident occurs sometimes in cases of active disease; in others the patient has been doing well and leading perhaps too active a life, when the stretched portion of lung gives way. The onset of the complication is marked by sudden pain in the side and high fever; and death often quickly follows from septic pyopneumothorax.

The following is a brief note of the case which came under our own observation:

B. E. P., aged twenty-four, shop assistant, was admitted into the Brompton Hospital on June 19, 1912, under the care of Dr. Hartley, suffering from pulmonary tuberculosis, of a year's duration, affecting the whole of the left lung and the upper part of the right. Tubercle bacilli were present in the sputum.

The case proved febrile, the temperature being irregularly raised, generally to 100° and sometimes higher. After some four months' treatment it became clear that the patient was not making progress, and an artificial pneumothorax was decided upon. This was performed by Dr. Hartley on November 5, the left pleura being punctured in the eighth space in the line of the angle of the scapula, and 200 c.c. of nitrogen, which gas we were at the time using, introduced. On November 7, 11, and 19, reinflations were given, 200, 375, and 300 c.c. of nitrogen being respectively introduced. The highest pleural pressure reached was +2; on the last occasion at the end of the inflation it was $-1\frac{1}{2}$. During this time the patient improved somewhat and the temperature was rather lower. The X-ray report by Dr. Melville showed a partial pneumothorax, the lung being adherent above the third rib, and in the region of the diaphragm.

On November 20 the patient experienced a sudden pain and feeling of "tightness" in the side, and the temperature rose. Two days later the pulse was 144 and the temperature 103°. The physical

signs of a partial pneumothorax persisted, as also the pyrexia, and the patient gradually became weaker. On December 18 she began to cough up purulent expectoration, amounting on one day to ℥xx., and this continued until death on December 23. Rupture of the lung into the cavity of the artificial pneumothorax, with pyopneumothorax and expectoration of the pus, was diagnosed, but the patient was too ill for surgical treatment.

At the autopsy a partial left pneumothorax was found, the lung being adherent over its upper third. A small perforation of the visceral pleura leading into the lung was discovered at the apex of the left lower lobe, and the pleura contained 4 ounces of pus. Two or three cavities, the size of a walnut, were present in the left upper lobe, with more recent infiltration of the lower lobe, and scattered areas of disease throughout the whole of the right lung.

Cases Suitable for Treatment.—From what we have said in the preceding paragraphs it is evident that treatment by artificial pneumothorax is not entirely devoid of risk, though modern methods of procedure have reduced this most materially. This being so, it must be agreed that *no case should be submitted to treatment until a full trial has been given to more ordinary methods.* If, however, the patient fails to respond to rest, sanatorium treatment, and medicinal remedies, then the question of an artificial pneumothorax should be considered, nor should this consideration be unduly delayed. The initial operation and early refills are often best carried out at a sanatorium, since here the patient has the hygienic and other advantages associated with such an institution.

The cases for which *the method is especially suitable are those in which the disease affects chiefly one lung,* so that when this is collapsed the diseased areas are at once brought under the influence of the treatment. In such patients, when other methods have failed, an artificial pneumothorax sometimes brings the temperature down in an almost dramatic manner, with a corresponding improvement in the patient's symptoms, although in our experience the fall is usually more gradual, a normal temperature being reached only after several inflations.

We have also seen benefit from the treatment in patients with chiefly unilateral disease of a less acute type than the above, but in whom any attempt to increase the exercise or work allowed leads to a set-back, whether indicated by a

rise of temperature, a rapid pulse, or increased cough and sputum.

The method is of great value also in the case of patients, generally with somewhat advanced disease, who are the subjects of repeated hæmoptysis, pointing to the presence of a pulmonary aneurism. In these cases the performance of an artificial pneumothorax will check the hæmorrhage, which may not recur, provided that the compression is duly maintained.

In cases of acute pneumonic phthisis the method holds out less hope, since here we are dealing with extensive and massive areas of caseation, yet in one such case we have known a gratifying result. We have also under observation at the present time a case of rapidly extending phthisis, not confined to one lung, with continuous high temperature, and in which the outlook appeared "hopeless," yet here compression of the most affected lung, though soon complicated by acute pleurisy with effusion, has led to great improvement in the patient's condition and a gradual fall of temperature to normal. Two somewhat similar instances are recorded by Professor Saugman.[4] Such cases are no doubt exceptional, and we would emphasise again that it is for *patients in whom the disease is chiefly confined to one lung or those whose life is endangered by repeated and uncontrollable hæmoptysis* that the treatment is especially suitable. Early laryngeal disease is no bar to the treatment, but it should not be advised in the case of patients with intestinal tuberculosis, weakened myocardium, or other serious complications.

Results of Treatment.—We have indicated that in suitable cases the immediate results obtained by the induction of an artificial pneumothorax are sometimes very satisfacory, the patient improving greatly during the continuance of the treatment, being enabled to lead an active life, and not infrequently losing his cough and phlegm, while tubercle bacilli disappear from the sputum.

With the cessation of the treatment these good results not seldom persist, the lung expanding to a greater or less degree, but the permanence or otherwise of the results will depend largely on the after-care. The patient must not regard himself as cured. Tubercle bacilli are still present in the lesions, and the disease will break out afresh if he be not

content to carry out, though in a modified manner, the mode of life which he has learnt at the sanatorium.

In other cases, though the immediate results may be encouraging, the disease in the other lung gradually extends and the patient finally succumbs. In others, again, the treatment fails, because the presence of adhesions prevents the due compression of the lung.

It will be interesting, in conclusion, to quote the results which have been obtained at the Berks and Bucks Sanatorium by Dr. Esther Carling[8] after eight years' experience of the method. The figures given below are brought up to date (June 30, 1920), Dr. Carling having courteously supplied us with the additional data, and verified the figures. The patients treated belonged to the working classes, and were all advanced or acute cases, with more or less unilateral disease. All had failed to respond to ordinary sanatorium treatment, and in all "the outlook was thoroughly bad."

Of the *54 cases* submitted to the treatment, in 12 it was not possible to effect a pneumothorax, and of these 12 patients 10 are now dead.

Of the *42* in whom compression of the lung was effected, 23 are dead. In more detail the results in these cases are as follows:

(*a*) In 12 the treatment failed and the disease progressed unchecked, until the death of the patient. This group, it should be noticed, included all the cases in which the lung was incompletely collapsed.

(*b*) In 8 temporary improvement was manifested. Of these 8, 2 are dead, and the remaining 6 are at home, but unable to work.

(*c*) In 22 marked improvement occurred. Nine of these patients have, however, since died. *The remaining 13 have returned to their old occupation, or to a moderately active and useful life.* Five of these 13 are continuing treatment by periodical return to the sanatorium or to the Tuberculosis Officer for reinflations.

Considering the type of case treated and the difficulties of efficient after-care in view of the class of patients dealt with, so marked and lasting an improvement in 13 out

of 42 patients must be regarded as a hopeful and encouraging achievement.

Surgical Intervention.

We have now to ask ourselves whether it is possible to assist in any way the 30 per cent. of cases which, though suitable on medical grounds for treatment by artificial pneumothorax, are yet unable to benefit from it owing to the presence of widespread adhesions.

Thoracoplasty.—For a few of them some form of extrapleural thoracoplasty may be considered, whereby varying lengths of the ribs (but not the entire ribs) from the first to the tenth or eleventh are removed on one side. The operation, which is infinitely more grave than that of artificial pneumothorax, is best done in two stages, so as to diminish the shock, and should be performed when possible under local anæsthesia, especial care being taken to anæsthetise the various intercostal nerves. With the avoidance of a general anæsthetic the risk of aspirating septic secretions into the healthy lung when the diseased lung collapses is greatly diminished, and the danger of the operation lessened.

We have known one or two notable examples of the successful performance of the operation, with great and lasting benefit to the patient; but the operation is an exhausting one, and even in skilled hands has a mortality of about 10 per cent. It should never, therefore, be contemplated until other methods of treatment have failed and until an artificial pneumothorax has proved impossible.

Professor Bull[9] of Christiania has recently published a record of 37 cases, of which 4 died as a result of the operation, and 11 are stated to have achieved a "curative result," by which is meant that the patients were at work, afebrile, and with the sputum no longer containing tubercle bacilli.

Professor Saugman[10] also reports the result of 40 cases treated at the Vejlefjord Sanatorium, 22 of which were operated upon by himself. Of these 40 patients, 4 died as a result of the operation, 12 are still in the sanatorium, and 13 are able to work, though of these 6 are capable of light work only. In regard to the type of case operated upon, 29 of the patients showed some degree of fever, but it is to be noted that the cases chosen for operation were for the most

part of the chronic type, with disease chiefly one-sided. Indeed, Professor Saugman expressly states that acute cases are not specially suitable, as so often active and progressive tuberculosis has already commenced in the other lung, which unilateral collapse will not check.

Rib Mobilisation.—Another operation which diminishes the volume of the chest and allows considerable collapse of the lung is that introduced by Wilms, and known as "rib mobilisation." We have referred to this when considering the treatment of bronchiectasis (p. 220), and need only add that it is an operation which, while having the same object and effect as thoracoplasty, is somewhat less hazardous, though by no means free from danger. It should therefore be considered when serious surgical intervention is contemplated, though no doubt the particular experience of the surgeon will be the important factor in deciding upon the exact method of operation.

In our opinion these drastic surgical procedures should only be considered in cases of pulmonary tuberculosis where all other methods, including artificial pneumothorax, have failed, and where the disease, if febrile, is not of a very acute type and chiefly restricted to one lung, or, if more chronic, keeps the patient a more or less permanent invalid. If the patient appreciates the deformity of chest which will result and the danger of the operation, and decides to take the risk, then the operation may be sanctioned, provided the heart and other organs are healthy and the general vitality of the patient sufficiently good. In our experience this favourable combination of circumstances is not often met with.

Opening and Drainage of Tuberculous Cavities.—The surgical procedures which we have found of value when dealing with abscess and gangrene of the lung (p. 360) are seldom profitable in the case of tuberculous cavities. Such cavities are rarely single, and are, moreover, usually situated at the apex or upper part of the lung, so that drainage *per vias naturales* is fairly well maintained, and the chief reason for interference is in the majority of cases not present.

Cases from time to time, however, do present themselves in which the extent of excavation, the superficial position of the cavity, and the large amount of secretion, with irritating and exhausting cough, suggest external drainage, and in

some instances an attempt is made by nature to find an external vent by perforation of the chest wall. If in such cases all other means have failed, and the patient's pitiable condition demands interference, opening and drainage may be considered.

An interesting case of this kind, under the care of Dr. John Hastings and Mr. Robert Storks,[11] was operated upon as long ago as 1844. The cavity, a large left apical one, was incised through the second intercostal space, and a drainage-tube inserted, with immediate relief to cough and expectoration. Since then other selected cases have been treated in a similar way,[12] and not infrequently with some amelioration of symptoms. But the operation cannot be looked upon as more than a palliative, and one which may alleviate the patient's most distressing symptoms, the harassing cough and abundant expectoration. Under the modern hygienic methods of treatment such large secreting cavities are not common, and intervention is now rarely called for.

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[3] "Apparatus for the Production of Artificial Pneumothorax," by S. Vere Pearson, M.D., *British Medical Journal,* 1913, vol. ii., p. 1098.

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[7] "Spontaneous Pneumothorax during the Course of Induced Pneumothorax," by M. I. Marshall and J. W. Craighead, *American Review of Tuberculosis,* 1917-1918, vol. i., p. 540.

[8] "The Value of Artificial Pneumothorax: Impressions after Eight Years and Fifty-four Cases," by Esther Carling, M.D., *Tubercle,* 1920. vol. i., p. 411.

[9] "Videre erfaringer om behardlingen av Lunge-tuberkulose med extra-pleural thorako-plastik," by Peter Bull (Christiania), *Norsk. Mag. for Laegevi-denskaben,* 1919, 80, p. 1105. (See *Tubercle,* 1920, vol. i., p. 330.)

[10] "Thoracoplasty in the Treatment of Pulmonary Tuberculosis," by Professor Chr. Saugman, *Tubercle,* 1920, vol. i., p. 305.

[11] "A Case of Tuberculous Excavation of the Left Lung treated by Perforation of the Cavity through the Walls of the Chest," by John Hastings, M.D., and Robert Storks, Esq., Surgeon, the *London Medical Gazette,* December 20, 1844.

[12] *The Surgery of the Chest,* by Stephen Paget, M.A., F.R.C.S., pp. 331 and 446. Bristol and London, 1896.

CHAPTER LII

TREATMENT OF PULMONARY TUBERCULOSIS—(*Concluded*)

Epitome.

It may be useful now to epitomise what we have said in regard to the treatment of pulmonary tuberculosis.

We have shown, in the first place, what hygienic and climatic measures may do, and it may be said that the limit of their efficacy has now been fairly ascertained.

We have further indicated what can be effected by the use of vaccines, sera, etc., and although these remedies have on the whole proved very disappointing, they have yet led us better to appreciate the mechanism of immunisation, and have enabled us to perceive that it is through the absorption of what may be called autogenous toxines, derived from lesions present in tuberculosis, that immunity is largely derived; and that duly regulated methods of hygienic exercises are the means of restraining such absorption within such limits as may bring about the end desired—immunity from further attack—and thus allow existing lesions to heal.

We have traversed the field of drug treatment, and have here, too, experienced many disappointments in the failure, time after time, of reputed specific medication. It must be noted, however, that amidst the débris of discarded remedies we may yet find encouragement for present hope and further research. For the treatment of the symptoms of the disease, cough, wasting, exhaustion, and special symptoms that may arise, we have many appropriate remedies. One class of drugs, also, seems to us to give promise of continued usefulness—namely, the antiseptic group of remedies, especially the creosote group, and perhaps also the derivatives of arsenic, as tending to check the activity of, if not to destroy, the *Bacillus tuberculosis* and its associated organisms, and to

render them more amenable to phagocytic and other attack.

Finally, we have seen that in artificial pneumothorax, whereby the lung is collapsed and placed at rest, we possess a method of treatment which is valuable in a certain proportion of cases when other methods have failed.

We may claim, therefore, that, since our last edition was published, progress in treatment has been made, and we must hope that the day is not far distant when research will place in our hands a specific means, whether medicinal or other, wherewith to combat the disease. This must be the aim of investigators, and we cannot believe that the hope is chimerical.

CHAPTER LIII

ON ABSCESS IN THE MEDIASTINUM

Suppurative mediastinitis is a somewhat rare disease, and is probably never of primary origin. The causes of abscess in this situation may be thus enumerated:

1. Gunshot wounds.[1]
2. Injury to the sternum.
3. Post-sternal syphilitic node.
4. Perforation of the œsophagus or injury from the impaction or penetration of foreign bodies, or malignant growth.
5. Septic causes, whether pyæmic in nature, or associated with enteric fever, or arising by extension from pneumonia, gangrene of the lung, streptothrix disease of the lung and pleura, or tracheotomy.
6. Glandular suppuration, most commonly in association with tuberculous disease, but sometimes occurring after whooping-cough.
7. Suppurating hydatid.
8. Caries of the spine.

Symptoms.—The presence of a wound, the history of a blow or of some ætiological factor of the kind alluded to in the above list, and the discovery of symptoms and signs pointing to mediastinal inflammation, are the elements of diagnosis in this often obscure disease. *Pain* and *tenderness*, *pressure signs* only very moderate in degree, and *pyrexia*, are the symptoms most worthy of notice. The pain is seated behind the sternum or between the shoulders, and radiates from these regions. Paroxysmal cough of the laryngeal type, a certain amount of obstruction to venous return, and some pain or difficulty in swallowing, are the pressure phenomena which may be observed in cases of abscess of considerable size. The pyrexia assumes the hectic type, and is attended with rigors and sweatings.

In abscess of the anterior mediastinum a certain degree of fulness over the superior sternal region may be noticed on inspection, and sometimes there is also a red blush over the surface, and a slight œdema masking the outlines of the cartilages and spaces. Some obscure impulse may be communicated from the aorta, and, in cases in which there is a large collection of pus behind the sternum, this impulse may closely simulate that of aneurism. There is dulness on percussion over the region of pain and swelling, with unduly conducted tracheal breath-sound. In cases in which the aortic sounds and impulse are conducted, it is important to make several examinations at times when the patient is at rest and free from cardiac excitement.

In posterior abscess there is often prominence and tenderness over one or two of the dorsal spinous processes. The percussion and auscultation signs are very obscure, so that the frequent association of spinal caries, with its characteristic external signs, is of much importance in diagnosis. X-ray examination may be of assistance by demonstrating vertebral disease, and defining the outline of the abscess.

If left alone, the abscess may point externally or rupture into the pleura, pericardium, lung or bronchus. In posterior cases it sometimes burrows along the vessels between the pillars of the diaphragm, and finally points in the iliac or femoral region. Most commonly anterior mediastinal abscesses either point externally or burst into a bronchus. Two cases which simulated abscess in the mediastinum, but which proved to be an aneurism and new growth respectively, will be found recorded in the succeeding chapter.

Treatment.—In the earlier stages fomentations should be employed, especially in those cases in which there is any external tenderness. In cases connected with caries of the spine the appropriate treatment for that affection must be adopted. Complete rest is in all cases necessary. The patient requires to be well supported by general treatment, and quinine may be administered for the control of hectic.

When the abscess is within reach of surgical treatment it should be evacuated. Cases, however, in which a communication with a bronchus has taken place should not be interfered with hastily, as the abscess may thus become completely evacuated and heal spontaneously.

Chronic mediastinitis and indurative mediastino-pericarditis require but little notice in this work, since they fall rather into the domain of cardiac diseases. The main symptoms, those of gradual heart failure, are chiefly due to the adherent pericardium which is so frequently present, although the picture is sometimes complicated by obstruction of the great venous trunks, owing to the cicatricial changes in the fibrous tissue in which they are embedded.

REFERENCE.

[1] For examples occurring in the recent war see *British Medical Journal*, 1916, vol. ii., Epit., Nos. 39 and 47.

CHAPTER LIV

INTRATHORACIC TUMOURS

TUMOURS within the chest may commence in the mediastinum and thence invade the lungs, or originate in the lungs and spread secondarily to the mediastinum. We shall, accordingly, consider the subject under the two headings: (1) mediastinal tumours, (2) tumours of the lung—according as the disease commences in one or other situation.

Tumours of the Mediastinum.

Various growths, both innocent and malignant, may be met with in the mediastinum. Of these the great majority are malignant in nature. Thus, Hare,[1] out of 520 published cases which he collected from 1830 onwards, found only 7 examples of fibroma, 11 of dermoid cyst, 8 of hydatid cyst (in most of which the mediastinal origin was doubtful), 3 of lipoma, 3 of enchondroma; and even these figures indicate, probably, too high a percentage, the uncommon cases having naturally found their way more readily into literature.

Of malignant tumours, the older statistics of Hare[1] and Wilson Fox[2] suggested carcinoma as the more frequent cause, but they were based largely upon cases of early date, when exact methods of examination were not at hand. Modern observations show that the new growth met with in the mediastinum is commonly sarcomatous in nature, and for the most part lympho-sarcomatous in type[3]; and this agrees with our own experience.[4] It is upon this form, therefore, the usual variety of mediastinal tumour, that our description of the disease will be based. To the simple tumours we do not propose to refer further. Apart from dermoid and hydatid cysts, which are described elsewhere in this work, these tumours, whether of the lung or mediastinum, are extremely rare, and only from their slow growth and less severe symp-

toms can they be distinguished from those of more malignant character.

Malignant growth may commence in various structures of the mediastinum. The usual seat of origin of sarcoma is in the lymph glands, either in those situated anteriorly in the upper part of the mediastinum (the superior mediastinum of anatomists), or posteriorly in the bronchial glands, or, more rarely, in the true posterior mediastinal glands. In a certain proportion of cases the growth originates in the remains of the thymus gland, and occasionally it commences in the periosteum of the vertebræ, or in the general connective tissue. When arising in the glands, the growth, having once broken through the capsule, tends rapidly to infiltrate the neighbouring parts, involving the pericardium and extending along the vessels and bronchi towards the root of the lung. Thence in the majority of cases it invades that organ (more commonly the right lung), spreading into its substance along the course of the bronchi, and eventually forming a large pinkish-white and fairly firm mass, often the size of a cricket-ball or even a child's head, which much distorts the natural shape of the parts. The appearance of the growth, with its radiating, bulbous prolongations, is illustrated in Plate XXXIV.

Carcinoma of the mediastinum is of rare occurrence. It sometimes occurs as an invasion from neighbouring parts, especially the œsophagus, or it may be secondary to growth in other organs, such as the stomach and liver. The mediastinum having become involved, the lung may be invaded later, as in the case of sarcoma.

Sarcomata of the mediastinum may occur at any period of life, from early childhood up to old age, but between the ages of twenty and fifty they are most common. Above fifty they are rare. The ages of those patients who died of this disease, and upon whom we have ourselves performed autopsies, were 19, 24, 26, 42, 45, 47, 48 and 62, respectively. Sarcomata are of more frequent occurrence in males than in females, although no satisfactory reason can be given for this curious fact. True carcinoma, on the other hand, rarely occurs before middle life and seems to attack the sexes equally.

Symptoms.—The symptoms of mediastinal growth are due to the presence of a foreign mass within the thorax, diminish-

PLATE XXXIV

SARCOMA OF THE MEDIASTINUM INVADING THE LUNG

The drawing shows the posterior portion of the right lung. The lower lobe is seen to be occupied by a large whitish mass of new growth, which originated in the bronchial glands, and spread thence into the right lung, especially the lower lobe, extending along the line of the bronchi by various tentacle-like processes. The growth, which proved on microscopical examination to be a lympho-sarcoma, had invaded and almost obliterated the right bronchus, and was also invading the pericardium and the wall of the right auricle. The right upper lobe was involved, but the left lung was free from growth. Secondary deposits were present in both suprarenals and in the left ovary. The right pleura showed some loose adhesions and contained a little fluid.

In addition to the sarcomatous disease, the apex of the right lung was the seat of recent tubercle. In the lower lobe was some acute broncho-pneumonia.

From a woman aged forty-two, who two months before her death was admitted into the Brompton Hospital with signs of right pleural effusion. Thirty ounces, and later twenty-six ounces, of serous fluid were withdrawn, and ten days before death ten ounces of blood-stained fluid. Death occurred after attacks of urgent dyspnœa.

(From the Museum of the Brompton Hospital. $\frac{7}{8}$ natural size.)

PLATE XXXIV

SARCOMA OF THE MEDIASTINUM INVADING THE LUNG.

To face p. 738.

ing breathing space, compressing vessels and nerves, and thrusting aside or invading important passages, organs and tissues. The first symptoms which usually attract the patient's attention are *cough* and gradually increasing *shortness of breath.* The cough, at first dry and teasing, or attended with slight and difficult muco-purulent expectoration, presents no special peculiarity; but later, when the growth compresses the trachea or bronchus, it acquires a peculiar "clanging" or "brassy" character, which the experienced ear at once detects. With complete paralysis of the vocal cord, following compression of the recurrent laryngeal nerve, the cough loses its explosive character and becomes husky or "bovine" in character. As the disease progresses, the expectoration becomes more abundant. When the growth has invaded the lung, the sputum may be sanguineous, and it sometimes resembles "red-currant jelly," the mucus coughed up being very intimately mixed with blood; but this latter form of sputum is by no means so essential to the disease as is sometimes supposed. In certain cases profuse hæmoptyis has been observed, an event sometimes preceded by the coughing up of fragments of the growth, although this is of unusual occurrence. In rare cases the hæmoptysis has proved fatal.

Pain in the chest is another early symptom of which the patient often complains. It is sometimes of the darting character peculiar to new growths, and, if the brachial plexus be involved, may not be restricted to the chest, but referred to the shoulder or radiate down the arm. More generally, however, there is a sense of oppression in the chest rather than of actual suffering, except in cases complicated with pleurisy.

The more distinctive *pressure symptoms* manifested by mediastinal growths arise from compression of the blood-vessels and lymphatics, the trachea and bronchi, the œsophagus and nerves. On anatomical grounds it follows that a tumour originating in the anterior mediastinum will compress the veins, while one originating in the bronchial glands will first compress the posterior mediastinal structures, giving rise to dyspnœa, laryngeal paralysis, ocular symptoms and dysphagia. The early pressure signs will therefore enable us in some degree to localise the position of the tumour. Towards the end of the disease, with the spread of the growth, the pressure symptoms become more general.

The growth affects differently the arteries and veins, the thick elastic walls of the former resisting its encroachment, whilst the thin walls of the veins are easily compressed and their lumen invaded, the mass sometimes appearing as a warty elevation within them. Inequality of the radial pulses is, accordingly, not often met with, whilst symptoms of compression of the superior vena cava or innominate vein, such as œdema, puffiness and lividity of the head, neck, and arms (especially the right side), together with enlargement of the veins over the upper part of the chest, are common. In aneurism, where the disease affects the arterial wall itself, the reverse holds good.

When the trachea or its main divisions are definitely compressed, cough and dyspnœa become very marked, the cough assuming the clanging or husky quality already described. Stridor also developes, and the attacks of dyspnœa acquire a most distressing paroxysmal character, in one of which the patient may succumb. These attacks are generally due to a plug of mucus accumulating near the point of compression and blocking the lumen of the tube.

Dysphagia is a symptom more often complained of in growths than in aneurism. The œsophagus, however, is so lax an organ that it may be stretched to a considerable extent over a mass of growth without any great difficulty in deglutition resulting, and, provided it be not invaded, it is rare for this symptom to give rise to serious trouble.

The recurrent laryngeal and the sympathetic are the nerves more commonly affected. Thus, abductor paralysis of one vocal cord, to be discovered only by largyngoscopic examination, and later complete paralysis, with weakening and hoarseness of the voice and husky cough, may be observed. This, however, is by no means so frequently present as in aneurism; indeed, we have on more than one occasion seen the nerve completely embedded and apparently lost in a mass of growth without its function being impaired. Ocular signs are more common. If the fibres of the sympathetic, which arise from the upper dorsal roots, be compressed and destroyed, the pupil will be unable to dilate, and will appear smaller than its fellow. Slight drooping of the eyelid, narrowing of the palpebral fissure, retraction of the eyeball, and interference with sweating on

the affected side of the head and neck, other well-known signs of sympathetic paralysis, are sometimes also observed. The interference with sweating may be easily demonstrated by dusting the part with powdered charcoal after an injection of pilocarpine. On the healthy side the powder adheres to the sweating surface; on the affected side it is easily blown away.[5]

Such are the chief symptoms which may be noticed in a case of mediastinal growth. Emaciation, excepting in those rare cases in which the œsophagus is early obstructed, is not a marked feature, although in all cases some loss of weight occurs. True cachexia is most uncommon. Pyrexia of a low and somewhat irregular type is often present; but in such cases the growth is rarely found after death to be uncomplicated, bronchiectasis or a slowly disintegrating form of septic broncho-pneumonia being most often discovered, and to these pathological changes, rather than to the growth itself, we must attribute the pyrexia, since in other cases fever may be entirely absent for weeks together.

Physical Signs.—The physical signs met with in a case of mediastinal growth will vary much, according to the extent of the disease. If the growth be recent, and confined merely to the glands, there may be no symptoms or definite signs, or at most some impairment of note over the manubrium sterni or the upper interscapular region behind. But as a rule distinct physical signs appear before death. We must note at once, however, that these signs are not necessarily due directly to the presence of the growth, but may be the result of pulmonary collapse, pneumonia, or bronchiectasis, with thickening of the lung texture, produced by the obstruction of bronchi by growth which has invaded their walls. The occurrence of a secondary effusion, either from pleurisy or compression of veins or lymphatics, often also complicates the physical signs. Bearing these facts in mind, we may now attempt to draw a clinical picture of a case of some little standing.

A slightly staring, suffused, and anxious expression of countenance is most commonly to be observed, and in marked cases the aspect assumes that of semi-strangulation, pitiable to witness, the swollen and œdematous head, neck and upper limbs contrasting with the natural appearance of the lower

half of the body. The respirations are quickened, the pulse generally somewhat accelerated, whilst the temperature remains normal, or is but slightly febrile. When the anterior portion of the mediastinum is involved, as is the case in many instances, some prominence of the upper sternum may be noticed. Enlargement of the glands at the root of the neck or in the axilla (see p. 86) should always be carefully sought for, this sign being of material assistance in diagnosis. The heart is displaced in a direction varying with the position of the growth. Most commonly it is simply thrust to one side, but sometimes the base is lowered, and the apex tilted upwards and outwards. At other times the growth extends from above downwards between the sternum and the heart, or, again, forwards from behind that organ; and we have met with an instance in which the heart was thus borne forwards, compressed and fluttering, against the anterior chest wall.

Over the region of the tumour there is percussion dulness, which, having been first observed in the area of the mediastinum, soon comes to encroach upon the limits of the lung. A careful percussion of the outlines of the dulness will in most cases reveal that the middle line of the chest has been transgressed—a fact of considerable diagnostic importance. A certain impulse communicated from the aorta may often be felt by the hand or appreciated by the stethoscope over the dull area; its knocking rather than expansile character can usually, but not always, be distinguished from that of aneurism. The heart sounds are well conducted by the growth, and a soft systolic murmur or souffle is occasionally to be heard over some portion of the dull region.

The respiratory sounds heard over the tumour differ according as the bronchus is still patent or is obstructed by the growth, which sooner or later penetrates the lumen of the tube. In the former case bronchial breathing, sometimes of an intense character, in other cases obscured by stridor, is heard over the dull area, and vocal resonance and possibly also fremitus are increased. If bronchial obstruction has occurred, then the respiratory murmur is enfeebled or annulled, and both vocal fremitus and resonance disappear. In such cases, on placing the hands evenly on the two sides over the lower regions of the chest, impairment of mobility

may be observed on the affected side during deep inspiration. Enfeeblement of breath-sounds over one lung is sometimes of diagnostic significance in cases in which there are as yet no other signs of mediastinal growth.

As the disease advances, in cases in which vital passages are not so immediately invaded as speedily to terminate life, the lung becomes more involved from root to periphery, until the whole side is completely dull. The chest may be distended from the pressure of the growth, but retraction of the side is by no means uncommon, either from shrinking of the growth itself or as the result of secondary collapse of the lung. Should the side be distended and the case be now observed for the first time, it may be impossible to distinguish it from one of extensive pleuritic effusion, for the heart is displaced, as it would be by fluid in the pleura, and vocal fremitus, resonance, and breath-sounds are all suppressed. The introduction of a fine trocar which fails to strike fluid, and is felt to penetrate and to be fixed by solid tissues, is the only means of effecting diagnosis, although careful examination will often reveal outlying islets of resonance corresponding with thin areas of lung encrusting the periphery of the growth.

An X-ray examination may help to elucidate the diagnosis, the growth being revealed as a non-pulsating shadow with somewhat irregular and ill-defined edge; but in other cases effusion into the pleura, either mechanical from pressure upon the azygos veins, or of inflammatory origin, complicates the signs and symptoms of the tumour, and obscures the X-ray picture.

Such effusions are often blood-stained, but this has no great significance, the fluid in cases of simple pleurisy being often of similar nature. Of more diagnostic importance is the cytological character of the fluid. In simple pleurisy with effusion a cytological count generally shows, as we have seen (p. 92), a predominance of small lymphocytes. In malignant disease small lymphocytes are also present in large numbers, and the count may closely resemble that of a simple pleural effusion. It sometimes happens, however, when the pleura itself is invaded, that there may also be observed cells of large size and irregular shape, often vacuolated in appearance, and with the nucleus pushed to one side, giving a

"signet-ring" appearance (see Plate XXXV.). If such cells are numerous and collected into groups, the picture presented is one very suggestive of malignancy.

A short account of the following cases will illustrate certain of the points emphasised in the preceding paragraphs:

Case I.—On March 27, 1904, one of us saw, in consultation with Sir Thomas Smith, and later with Sir A. Pearce Gould and Dr. Whittick, a lady, aged forty-eight, who complained of painful and difficult deglutition, with radiating pains about the sternum and to the shoulders. The history of her illness, rather obscure and indefinite, was as follows:

The lady was of active habits, a good and fearless rider, and, except for some severe hunting accidents, had enjoyed excellent health. Two years before she had a bad fall from her horse, but made a good recovery. In November, 1903, she suffered from influenza, followed by pains below the left breast, round the chest, and down the back and leg, which were regarded as due to influenzal neuritis. In February, 1904, she consulted her physician for symptoms of acid dyspepsia, with occasional slight pyrexia (100°), gastric pains and intercostal tenderness. There was pyorrhœal affection of the gums, and the chest and shoulder pains were attributed to "a septic infection from alveolar pyorrhœa irritating the fibrous tissues of the pleura and œsophagus." Sufficient importance was, perhaps, not attached to the difficulty in swallowing, or, rather, the sense of constriction opposite the third piece of the sternum, attended with pain of a radiating character, which had existed for some weeks, until it amounted to an obstruction, and which, on Sir Thomas Smith's first seeing the patient on March 26, rendered partial feeding by the rectum necessary. At this time a short cough had supervened, and there was occasional retching with the removal of mucus, slightly tinged with blood and containing some purulent opacities.

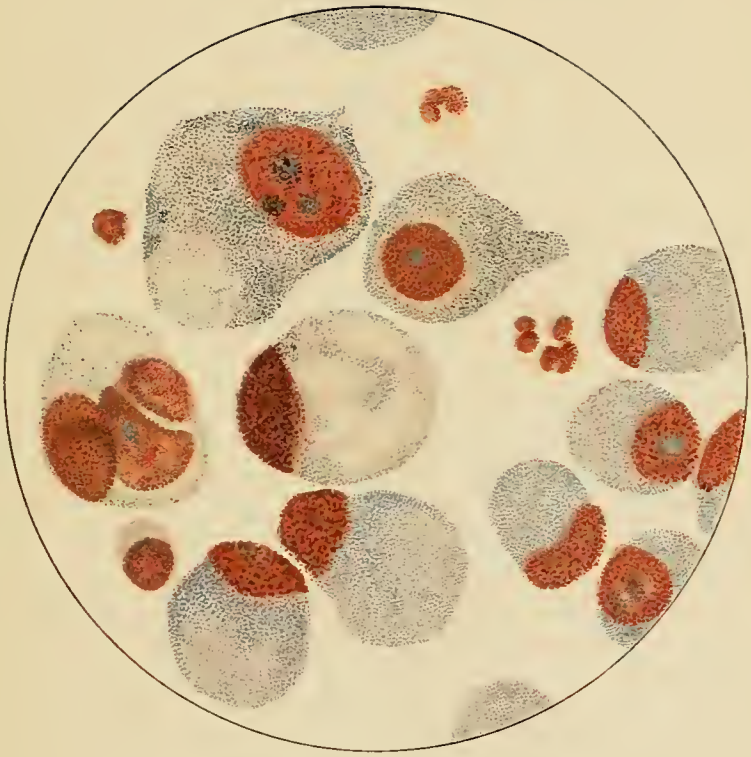
On March 27 the following conditions were noted: The patient had passed a restful night owing to morphia, but there had been slight retching of lightly stained mucus. The countenance was pale, with no marked anxiety of feature; respirations quiet, although slightly quickened; breath notably fœtid. The pulse was small and compressible (she had been fed by enemata for the last sixteen hours). There was also occasional slight cough with expectoration of mucus containing a few pus and blood corpuscles. On giving her a little milk to drink, an evident difficulty in the passage was experienced at about the junction of the upper and middle third of the œsophagus, and some pain was complained of in the effort of swallowing. The milk was not, however, returned. Over an area corresponding with the upper portion of the sternum to the level of the third cartilage, and extending to the left for an inch beyond the

PLATE XXXV

CELLS FROM A CASE OF PLEURAL EFFUSION OF MALIGNANT ORIGIN

The cells were obtained by centrifugalising the fluid removed from the pleura during life. From a female patient, aged forty-seven, who died from new growth of the lung and pleura. The large size of the cells, their irregular shape, dropsical appearance, and the expression of the nucleus to one side, producing a "signet-ring" appearance, suggested the presence of malignant growth, which was confirmed at the autopsy.

(Drawing by Dr. I. C. Maclean, from a preparation by Dr. A. C. Inman; stained by Giemsa's modification of Romanowsky's stain. × 750.)



Cells from a case of Pleural effusion of malignant origin.

To face page 744

sternal margin, there was dulness, and, on application of the hand, a slightly heaving impulse, which was more distinctly appreciated by the finger-tips pressed into the interspaces at the left sternal margin. The expansile impulse was still more evident to the ear, using the solid stethoscope, and with each impulse there was a *bruit de souffle* closely resembling in character the placental murmur. The heart's position was normal, and there were no altered cardiac sounds. The respiration over the left front was weak and sub-bronchial in quality, and generally over the left side posteriorly the percussion note was less full and the respiration weakened, becoming in the lower interscapular region somewhat bronchial in quality and accompanied by a few crepitations. On the right side, both in front and behind, the respiratory sounds were exaggerated.

The diagnosis arrived at was that there was a soft and rapidly growing and very vascular tumour in the anterior mediastinum, pressing upon and more or less involving the œsophagus, and commencing to invade the rest of the lung. In the male subject an aneurism, especially of the dissecting kind, might have been more seriously in question, particularly with the history of a severe hunting accident; but there were no signs of alteration of the cardio-vascular system and no accentuation of, or murmur with, the second sound of the heart. The possibility of a pulsating mediastinal abscess had more carefully to be considered; and, although the bruit seemed to be so distinctly intrinsic to the tumour, and there was an absence of any marked excursion of temperature or hectic symptoms, still, the possibility could not altogether be ignored, and the patient volunteered the statement, without any suggestive questions, that she felt a throbbing as of an abscess within her chest.

It was agreed that some exploration should be made, and Sir A. Pearce Gould was asked to see her on the following morning with this intention. Meanwhile Dr. Hugh Walsham was requested to take a skiagram, from which the evidence of the conditions being due to growth, not fluid, was strengthened. On March 29, after careful consultation, it was decided that the safer, as well as the more satisfactory, plan was to raise the sternal end of the pectoral muscle, and make an incision through the intercostal space. An anæsthetic was given, and the sternal attachment of the muscle having been divided, the second cartilage was exposed, and on cutting through the space it was at once obvious that a soft growth was underneath. The second cartilage was excised with a view to relieve pressure, which enabled the growth to be further examined, and the wound was then closed and stitched up. The structure of a fragment of the growth removed proved on examination to be that of a round-celled sarcoma, extremely vascular and presenting numerous hæmorrhagic extravasations. Some decided temporary relief was afforded by the removal of the cartilage, and the consequent diminution of internal pressure, and the lady took some food afterwards with but little difficulty. She died, however, nine days after the operation.

Case II.—The following case shows the disease commencing with the features of simple pleural effusion, as so often happens:

Mrs. T., aged fifty-six, was seen with Dr. Farr on November 5, 1909. She had suffered from influenza twelve months previously, and had been ailing since. In the previous August she went to Llandrindod. On October 29 the respirations were stated to have been 28, and some friction was heard over the pericardium. At the time of the consultation on November 5 there was a paroxysmal cough, but no expectoration; the temperature was 99°; the urine phosphatic. On examination the right side of the chest was found to be dull over the anterior base, and the left dull to the angle of the scapula.

On November 10 the dulness had extended upwards on the left side. Skodaic resonance was elicited below the clavicle, and over the upper confines of dulness behind at the mid-scapular region the breathing was tubular; the dypsnœa was considerable. The physical signs thus far pointed to simple effusion, and a pint of fluid was withdrawn, tinged with blood, which it was thought might be due to the rupture of a small vessel during the operation.

By November 20 the effusion had again increased up to the clavicle, and grave doubts were now expressed as to growth being in the background, owing to the slightness of the febrile reaction, the insidious onset of the illness, and the blood-stained fluid. A further tapping was recommended. This resulted in the withdrawal of but twelve ounces of fluid, which, according to the report of the Clinical Research Association, "contained a moderate amount of blood and a small clot, also coagulated lymph, and many small masses composed of cells of variable size and more or less irregular shape. Cells mostly rounded, much vacuolated, and degenerated. No pus cells, tubercle bacilli, or other organisms were found. The characters of the fluid are almost pathognomonic of growth." Dr. Farr added, in sending the report: "I think the growth has much increased; she is now so much distressed with urgent dyspnœa that I have commenced morphia injection." The patient died shortly after.

Case III.—The following case was one in which the diagnosis of mediastinal growth emerged through that of emphysema and dilated heart, from which the patient had for ten years been known to suffer:

Mr. O., aged forty-eight, was seen with Sir Malcolm Morris in October, 1905, the patient having been under observation since 1896 for emphysema. In November, 1905, he was sent to Torquay, where he became less well, with increasing difficulty of breathing, especially at night; he also had some attacks of dark hæmoptysis of not more than half an ounce in amount. At the end of August, 1906, he was again seen, and the signs observed were those of general emphysema,

but with less fulness of note over the right side, and, over the lowest three ribs posteriorly, dulness, regarded as due to collapse of lung from pressure of a somewhat enlarged liver, which was palpable for two fingers' breadth below the costal margin. The right side of the heart was dilated, and a systolic murmur was audible at the apex.

On November 8, 1906, he was again examined with Sir Malcolm Morris, when a notable absence of breath-sound was observed over the right side of the chest, and some dulness was noted in the right interscapular region. The first diagnosis of emphysema, mitral incompetence, dilated heart, and pulmonary infarction, was now supplemented by that of posterior mediastinal growth to which the hæmorrhagic attacks were attributable. The disease made rapid progress to a fatal issue. The temperature hovered between the normal and 99°.

Case IV.—The following case, one of aneurism of the transverse aorta, presented many of the features of mediastinal growth, or possibly of abscess behind the sternum, and therefore may perhaps be usefully introduced here:

Captain C., aged fifty, was seen with Dr. Humphrey on March 4, 1897. There was a history of syphilis, and he had led physically a strenuous life. In February, 1895, he had laryngitis, and suffered for twelve months from some asthmatic symptoms. In September of that year he coughed up three-quarters of a pint of pus, and since then had had eight similar attacks, the last in December, 1896. The expectoration had been purulent since then, but not fœtid, and occasionally more purely bronchial mucus had been expectorated. Of late he could only sleep in the sitting posture. His weight had diminished from 11 stone to 9 stone 12 pounds.

The features were congested to turgescence, and there was very considerable distress in breathing. Over the manubrium sterni, and a little to right and left of it, there was dulness, and pulsation was felt over a small area corresponding with the inner end of the second cartilage and left space. Posteriorly, in the interscapular region on either side and over the spine as far as the fourth process the breath-sounds were notably bronchial.

As regards the diagnosis in this case there was some difference of view, that which appeared to us most probable being that of a sacculated aneurism presenting from the back part of the transverse aorta, pressing upon the lower trachea, and causing muco-purulent secretion to accumulate behind it; the other view being that there was an abscess or growth in the upper substernal region connected with the bronchus, and having pulsation communicated to it from the vessel. This latter view was favoured by the report of an X-ray examination.

The patient's symptoms becoming aggravated, it was decided, in consultation with Sir Alfred Pearce Gould, to explore the upper medi-

astinum, and on March 30, 1897, Sir Alfred trephined the manubrium sterni, and dissected down to the aorta. "No abscess or swelling was detected; the aorta appeared very large, and pulsation was felt all round the finger." The wound was then closed. On April 10 the patient went to Brighton. "The operation did neither good nor harm; if anything, he was more comfortable afterwards." From a report kindly furnished by Dr. Halstead, who had the care of Captain C. for some time before the operation and subsequently at Ramsgate, it appeared that there was no discharge of pus after the operation, with the exception of a few drachms in July, until about a fortnight before his death, during which time Captain C. brought up daily approximately four ounces of muco-pus and suffered much distress. He died from exhaustion on September 26, 1897.

Diagnosis.—The diagnosis of tumour of the mediastinum, as soon as symptoms seriously draw attention to the case, is not as a rule difficult, since pressure signs and symptoms are incompatible with any other form of pleuritic disease or pulmonary consolidation, nor are they present in any decided degree even in extreme pericardial effusion.

The conditions which most simulate tumours in this region are abscess of the mediastinum and syphilitic stricture of a main bronchus. With abscess the temperature, hectic phenomena, possible history of injury, the character of the pain, and the attendant inflammatory phenomena, will usually clear up the difficulty, although we have already described a case in which abscess decidedly entered into the question of diagnosis, and another case in which the diagnosis between an abscess beneath the sternum and an aneurism was only decided in favour of the latter, and contrary to the interpretation of an X-ray examination, after a portion of the sternum had been trephined. The distinction of syphilitic stricture of the main bronchus or lower trachea from an obscure mediastinal tumour of small dimensions strangulating these parts is most difficult. Still, the limitation of pressure signs to one system and the absence of any discoverable signs of tumour, together with a history of syphilis, should at least aid the diagnosis sufficiently to suggest a definite treatment, the effects of which may, perhaps, though not necessarily, throw further light upon the case.

Having excluded mediastinal abscess and syphilitic stricture, and arrived at the conclusion that a tumour is present, we have yet to determine whether it be aneurism or growth.

In favour of growth the following are the more important points: (1) the age of the patient (if below twenty-five); (2) the presence of an extensive area of superficial dulness; (3) the presence of decided venous obstruction; (4) the absence of marked disease of the arteries, of characteristic pulsation, diastolic shock, tracheal tugging, and a history of syphilis; and (5) the history or presence of tumours in other situations. In a doubtful case an X-ray examination should always be made. With growth we see a shadow, which in early cases, before pulmonary and pleural complications have set in, fades gradually at the edges, and presents no clear evidence of pulsation; with aneurism we may perceive a tumour clearly defined in shape, and evincing definite expansile pulsation, a combination which, when clearly visible, is very characteristic.

The diagnosis of new growth from pleural effusion, which is sometimes so difficult, must be settled, as we have seen, by the exploring syringe; but we must remember that the two are not infrequently combined. In such cases a microscopic examination of the cells present in the fluid (see p. 743) may prove of value, as in one of the cases which we have just recorded.

With regard to the nature of the growth, it is to be remembered that, if the disease be primary in the mediastinum, it will probably be a sarcoma, and the younger the patient, the more likely is this to prove the case. Should the disease be secondary to a growth elsewhere, it will be of the same nature as the primary disease.

Prognosis.—Malignant disease of the mediastinum is inevitably fatal, but its duration varies much. Of sixty cases occurring at the Brompton Hospital, investigated by Dr. J. N. MacBean Ross,[3] the average duration of life from the date of the first symptom complained of was thirty-two weeks, the maximum duration being eighty-eight weeks, and the minimum nine. Death is generally the result either of gradual exhaustion or of suffocation, the patient in the latter case dying after prolonged attacks of dyspnœa. Less commonly it is due to hæmoptysis, sudden syncope, or cerebral tumour. At the post-mortem the disease may be found restricted to the mediastinum and lung, but it is more common to discover secondary growths elsewhere, the liver, pan-

creas, suprarenals and kidneys being the organs most commonly attacked.

Treatment.—The cases of mediastinal new growth which we have been considering are beyond the reach of surgery, and their progress is not influenced by drug treatment. *X-rays* and *radium* are not capable of arresting the disease, but we believe that in more than one instance we have seen the progress of the malady checked and the fatal issue somewhat postponed by the use of one or other of these remedial agents. We accordingly as a rule advise in these cases a course of radium or X-ray treatment.

The more distressing symptoms complained of, including the paroxysms of dyspnœa, must be relieved by sedative remedies. A combination which we have used with considerable advantage, at least for a time, is one of iodide of sodium, from three to five grains, and chloral, five to ten grains, taken four or six times in the twenty-four hours. Morphia, in combination with atropine, must also be employed and pushed if necessary. Temporary relief to the dyspnœa may also be given by oxygen. Should an effusion into the pleura occur, it must be aspirated, if it definitely increases the difficulty of breathing; otherwise it is best left alone, for it does no harm, and if removed, it will almost certainly recur.

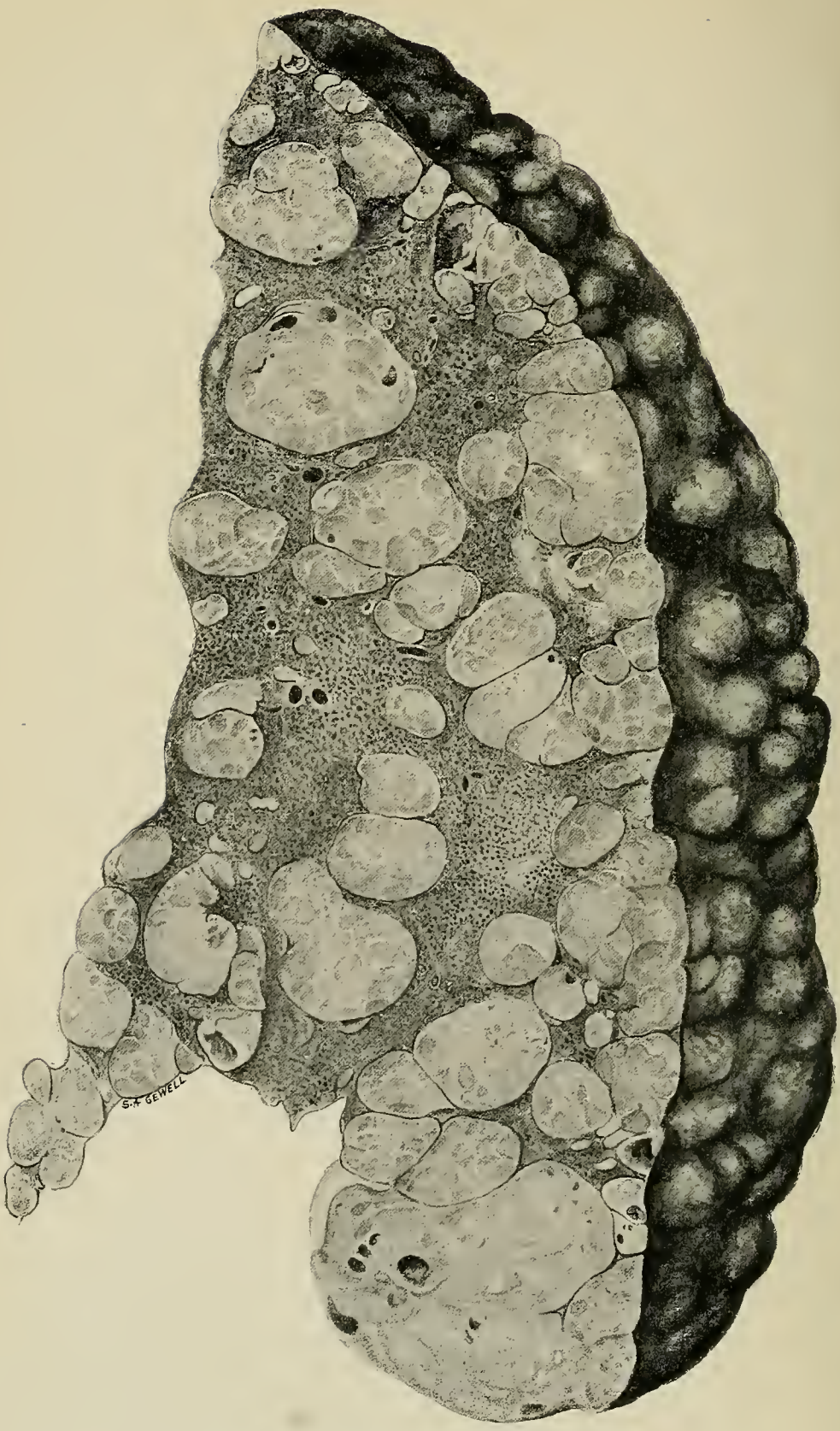
Tumours of the Lungs.

In the description which we have just given of mediastinal tumours we have shown that in the great majority of cases, before death occurs, the disease spreads into the lung, and forms there a mass of growth, often of very considerable size. We have now to consider those tumours which may affect the lung irrespective of the mediastinum, or only spread into the latter at a later date.

Tumours of the lung proper, like those of the mediastinum, may be innocent or malignant. Among the former, fibromata, lipomata, and others, have been described; but they are all so excessively rare, grow so slowly, and cause so few symptoms, that the group possesses no clinical importance, and we shall not again refer to them. Hydatids, dermoid growths, and gummata we have described elsewhere.

Primary Carcinoma of the lung is not so rare as is com-

PLATE XXXVI



SECONDARY CHONDRO-CARCINOMA OF LUNG.

To face p. 751.

monly supposed, Dr. Adler[6a] having recently collected 374 examples. The disease, like mediastinal new growth, is more common in males than in females, and usually occurs in patients over the age of forty. It originates most commonly in the epithelium of the bronchi, more rarely from the cells lining the alveoli, and gives rise either to a large mass within the lung, or, as in a case which we have recorded,[4] to an infiltration of a portion of the organ. In other cases the growth commences in the bronchi near the root of the lung and soon gives rise to symptoms of compression. In *secondary cases*, which are often met with, the tumours generally form numerous separate masses, scattered throughout both lungs in a fairly symmetrical manner, and on section varying in size from that of a threepenny-piece to a shilling or even larger (Plate XXXVI.). In rare cases the secondary growths may be extremely small and very numerous, so as to resemble miliary tubercles, and to this condition the name "carcinomatosis" has been applied.

Primary sarcoma originating in the lung is undoubtedly very rare, most cases which seem at first to suggest this origin proving on investigation to be examples of mediastinal disease, with subsequent invasion of the organ. Nevertheless, true examples occur from time to time. In such cases the growth forms as a rule a solid mass, which more or less replaces the lung tissue of a whole lobe, whilst the bronchial and mediastinal glands remain unaffected, or show signs merely of recent invasion. *Secondary sarcomata*, on the other hand, are common, and they, too, like secondary carcinomata, are generally multiple and bilateral. Occasionally, however, they take the form of single massive tumours, which give rise to all the symptoms of pleural effusion. Of the large displacing tumours, osteo- or chondro-sarcomata are the most common, and are generally secondary to affections of bone or joints.

Symptoms.—Primary malignant disease of the lung presents symptoms which closely resemble those which we have seen to occur in mediastinal new growth. Cough and shortness of breath, often paroxysmal in character, and pain in the side, are commonly complained of; emaciation is not a marked feature, and pyrexia in uncomplicated cases is usually absent. The sputum is from time to time blood-stained, and decided

hæmoptysis may occur. Symptoms of venous compression and pressure on the nerves and large air-tracts are, however, less frequent and longer delayed than in ordinary mediastinal tumours, although, if the patient survive and the mediastinum become affected, they may make their appearance.

The physical signs, again, are in no sense characteristic. Over the portion of lung affected impaired note, weak breathing, and diminished vocal vibrations, or in other cases bronchial breath-sounds and bronchophony, according as the bronchus is obstructed or free, will be the signs elicited. In other words, we have a gradual consolidation of the lung, usually of the lower lobe, proceeding from above downwards, with, in most cases, weakened breath-sounds and hæmorrhagic sputum. An X-ray examination, provided it be undertaken early in the course of the case and that there is no effusion into the pleura, discloses the shadow of the tumour, somewhat irregular in outline, and often with an ill-defined edge. Later, with associated collapse of the lung and other pulmonary and pleural changes, the X-ray picture becomes obscured.

Secondary growths in the lungs from a primary focus in some distant organ, even when numerous and scattered through both lungs, not uncommonly remain latent, the condition being first discovered on the post-mortem table. When, however, the growth forms, as it occasionally does, a large single mass, physical signs often suggesting a pleural effusion will make their appearance.

In duration and mode of death cases of primary malignant growth in the lung do not differ materially from those in which the mediastinum is first attacked. Eight to ten months is the average duration of the disease; gradual asthenia and suffocation the common precursors of death.

Treatment will be similar to that for mediastinal growth (p. 750). In cases diagnosed quite early, in which the disease appears localised and the mediastinum not as yet involved, surgical intervention has been essayed in a few cases, the tumour and affected lobe of the lung being removed. In one case[66] the patient's life was apparently somewhat prolonged, but the operation is so grave and hazardous that there can be but very few cases for which it would be suitable.

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